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The Arthur E. Mills Memorial Oration.[1]

AUSTRALIA'S EMPTY SPACES.

By FRANK J. S. WISE, M.L.C.

I DEEPLY APPRECIATE the privilege and honour of being invited to deliver the Arthur E. Mills Memorial Oration.

The commemoration of the name of Arthur Edward Mills in the manner established by The Royal Australasian College of Physicians keeps attention focused on the outstanding and inspiring contribution this distinguished man made to human welfare. In addition, and in the manner he would have chosen, the opportunity is offered annually to provoke thought in the promotion and encouragement of medical education and general culture and the dissemination of knowledge.

In an endeavour to provoke thought and to assist in the dissemination of knowledge, I have chosen the title "Australia's Empty Spaces" as the basis of my remarks to you.

[1]Delivered on May 2, 1961, at the Annual Ceremony of The Royal Australasian College of Physicians in the Winthrop Hall of the University of Western Australia.

The theme makes a fascinating study, and to assist in stimulating interest in it I have had prepared a map which will present to the eye the vastness of the sparsely settled areas of Australia.

How empty is Australia? How can we fill the empty spaces? Is closer settlement practicable in the empty areas? Since it is a dry continent, what is the solution of the water problem? What latent wealth may be in our empty lands awaiting development in cattle and sheep production, in agriculture and in mining? What is the marine wealth adjacent to our shores? Are there health difficulties in promoting settlement? What of transport costs and high taxation?

By some world standards, and judged on our own capacity further to develop the resources of the more densely settled areas, including all Victoria and Tasmania, our peopled areas are considerably under-populated. It may be said realistically and safely that our peopled areas could have a population increase of 10 millions, while the sparse lands have difficulty in adding one million. In the more pleasant places to live the population increase will be easy to achieve.

In the sparsely populated regions are vast areas of our pastoral lands, which carry tens of millions of sheep, many millions of cattle and relatively few people who, in the aggregate, produce enormous exportable wealth. Quite

apart from economic considerations I think our concern should be to review all that is known of our empty land, to learn all we can about it, to assess what we may do with it and to develop a national policy for it.

For the purpose of a somewhat cursory analysis we may use as a starting point the glaring fact that 2,200,000 square miles of Australia (74·7% of the total area) carries but 1·9% of the population. In approximate figures, over 98% of Australia's people reside in a quarter of the area of the continent.

Bad as that figure may appear, a map could be drawn showing 45% of Australia which has one quarter of 1% of our population.

New South Wales is generally regarded as a somewhat densely populated State, but the fact remains that 40% of New South Wales has 1·7% of its population, or only 65,000 people in that area which includes Broken Hill.

South Australia has 1·1% of its population in 78·7% of the area, and Western Australia has but one person to each 26 square miles in 86·2% of its total area—32,000 people in 841,000 square miles.

When it is considered that many important towns such as Broken Hill, several large pastoral towns of Queensland, Darwin, Alice Springs, Wittenoom and Yampi are included in the 74·7% carrying 1·9% of Australia's population, a thought-provoking commencement for our theme has been raised. Very striking for us and perhaps more striking still for the uninformed foreigner is the figure that in all Australia's three million square miles the density is but 3·4 persons per square mile, whereas Europe has a density of about 100 per square mile. There have been many suggestions as to the future population of Australia. Since Australia and the United States of America have a similar area, some people have expressed the view that Australia must be able to support 150 millions of white people when we have been developed as long as the United States of America.

Sometimes the unrealistic investigator has fitted most of the countries of Europe into the map of Australia to prove that Australia should support 400 million people. Be that as it may, emptiness is obviously provocative to other nations, whatever may be the facts about resources. It has been said that 1200 million pairs of eyes of residents of countries to our north are anxiously looking at empty northern Australia.

It is very necessary for all loyal and thoughtful citizens of Australia to settle down in thought and to assist in dispelling many foolish notions in regard to our empty spaces and what may be done with them. There is no merit in ignoring the truth and no advantage in not making it widely known. It is important to take stock of the situation. It must be borne in mind that Australia has the second largest area of arid land in the world, that it is the driest of all the continents and that in its problem areas there is a variation in land from absolute desert, with a sparse and indefinite rainfall which so far has rendered it incapable of utilization by permanent pastoralists, to the small higher-rainfall areas of the north-western, northern, and north-eastern coastlines, which have a rainfall of from 50 to 150 inches per annum.

Australia has at least half a million square miles of desert which may not be capable of producing anything other than minerals. Our limitations may be the absence of edible plants. The Sahara desert contains 2·6 million square miles and carries a million people and millions of head of stock.

It is important that the world should know that much of our continent is destined to remain empty because of the unlikelihood of it ever being able to sustain life from natural forage.

The greatest limiting factor in the future population of Australia is likely to be the availability of water. There are few parts of Australia, even in the heaviest and most reliable rainfall areas, where the provision of water to meet the demands of a growing population and expanding industry is not a current problem. Surveys of our water resources, namely the major river systems of the continent, are up to date and there is a continuing examination by experts of the sources of supply of water for our cities and towns. Side by side with the pursuit of that problem are the energies being devoted to the supplying of water from a variety of sources for our extensive flocks and herds.

An intensification of the search for a means of removing salt from sea water, from salt lakes and from saline or brackish ground water, is vital to the continuing occupation of much rural land, and to the expansion of sparse-land occupation. In a few places in the world, such as the coastal areas of south-western Africa and the island of Curaçao, fresh water is obtained from sea water to supply whole communities. Lack of new sources of fresh water will soon limit further expansion and production in some populated areas and restrict land use in remote places. Desalting of water is a very important matter of world research and of the greatest moment to us. No greater contribution in the lifetime of any one scientist could be made than to make fresh water available from the sea, or from other saline waters at economic rates. Availability of fresh water for stock and human use, for mining purposes and for processes in industry could be revolutionary in Australian progress. It would be a milestone in world history and could revolutionize the use of land in Australia's empty spaces.

In our sparse lands are a few permanent rivers of short watersheds over 1500 miles of coastline, and enormous areas where rivers are absent or flow but rarely.

A big proportion of our continent has an annual average rainfall of under 10 inches. Of all the varied climatic elements affecting effective occupation the decisive one is rainfall. The season of the rain and the regularity of its occurrence are matters as important as the total amount. Annual average rainfall can give only a very approximate idea of the possibilities of settlement. Rainfalls of any specified volume are not of the same value in different places. Tennant Creek in Northern Territory, Roebourne in the north-west and Northam in Western Australia all have about 15 inches annual average rainfall. Tennant Creek has a totally dry period of seven months, and is in an arid climate of high temperatures; Roebourne's rainfall has varied from 13 points to 42 inches in different years, and is in sparse pastoral country producing good-quality wool; Northam is in a wealthy and important safe wheat-farming district of reliable rainfall, most of which coincides with the wheat-growing season.

In the semi-arid areas of unreliable and ineffective annual rainfalls, an opulent season can be very misleading as an indicator of settlement prospects or of carrying capacity. After a heavy wet season the appearance of the country does not resemble in the least its appearance in nine years out of ten. The Nullabor Plain sometimes looks like a wheatfield. It is interesting to observe that there has been very little penetration by pastoralists of the areas unoccupied for stock raising when Warburton crossed the arid north of the continent, from east to west, nearly 90 years ago. Emphasis is given to the fact that the best of our arid and semi-arid lands have been occupied by pastoralists and have been productive for a long time, and that, with the exception of the country lying between Alice Springs and Halls Creek, most of the balance may be destined to remain empty for pastoral production, mainly because of the lack of suitable food for stock. Robert Kleberg of King Ranch, Texas, and now also of Brunette Downs in the Northern Territory, is one qualified to speak on the cattle industry with authority. He has said: "I feel that in the cattle world the emphasis should be on the breeding of beef cattle adapted to unfavourable environments and believe further that the future expansion of large-scale beef production will be in the wet and dry tropics in the semi-desert areas of the world." If one assumes Mr. Kleberg to be right, the central and northern part of Australia is one of the world's great areas for increasing cattle production. Some of Australia's most reliable authorities on the subjects of wool industry and sheep husbandry strongly hold to the view that large

expansion in wool production in the pastoral—as distinct from the farming—areas is extremely unlikely.

What then of the enormous area devoted to cattle rising in northern and central, eastern and western Australia, included in our sparse lands, which would be roughly 750,000 square miles in extent? In this area there are now at least six million cattle. One-third may be said to be good cattle country, notably the Channel country and the Victoria River region of the Kimberleys, one-third fair cattle country and one-third inferior to worthless for pastoral purposes, the better-quality land being distributed through the whole area. Over the whole of this country the same variations in rainfall exist, while offers enormous scope and is a prospect of the highest order and importance in our economy.

Road development right across north Australia is a vital national need for the development of pastoral prospects, both existing and latent. Considerable capital expenditure is warranted as a national plan, and large sums of private spending are involved.

In regard to agriculture, it may be said that for many reasons, which are obvious from my earlier comments, it is difficult to foresee vast economic agricultural expansion in the area under review. We are seeking greater population centres than are compatible with pastoral pursuits,

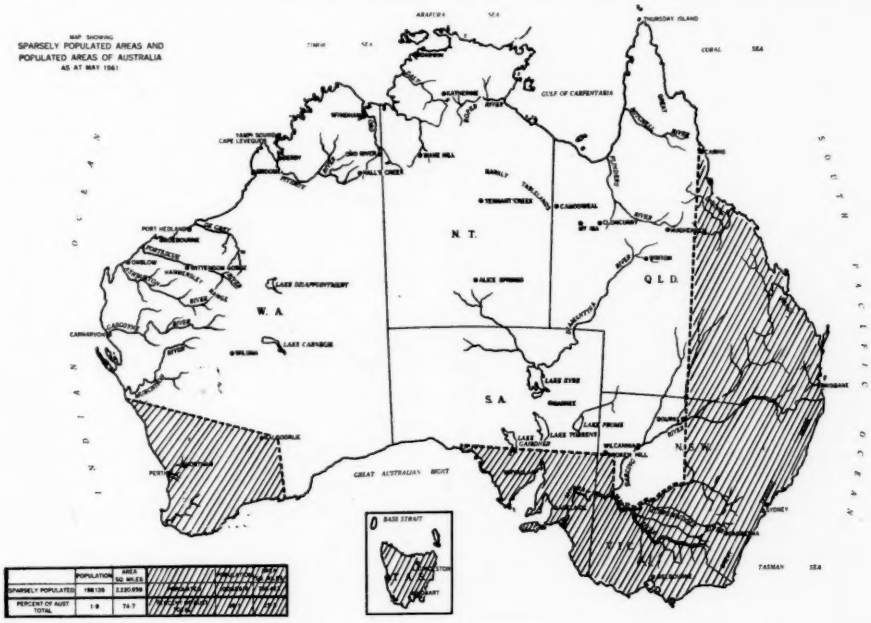

FIGURE I.
Map showing populated areas and sparsely-populated areas of Australia as at May, 1961.
Hatched areas indicate populated areas.

the same climate, the same sparseness of population, the same shortage of experienced cattlemen and the same heavy transport costs obtain. There is very little difference in the taxation laws applying to residents of Perth, Alice Springs or Halls Creek.

Many meatworks have been built and abandoned through the years in northern Australia, and more will operate in the years to come, but for the time being Wyndham stands out as the outpost asset with its annual kill of between 30,000 and 40,000 head. It is likely, too, that more air beef centres will be developed. One of the great prospects is in the area where 100,000 buffaloes now graze, producing little or no beef and a few hides.

The coastal plains from Victoria River to the Gulf of Carpentaria await a national project of development which may consist of the replacement of the buffalo by cattle bred for difficult environments. The field is wide open for the geneticists — men of the type of Dr. J. Rhoad of King Ranch fame, and Dr. Bousma of Pretoria, who have evolved and proved new breeds of cattle for unfavourable environments. For the pastoral industry urgent needs are the lessening of production costs including taxation, thousands of miles of fencing, hundreds of water points, better animal husbandry, better transport and the production of a better quality beef. Implicit in it all is intensive veterinary research. A plan for 20 years in developing new breeds of stock for difficult environments but for more than 40 years, much energy and large sums of money have been expended in endeavours to promote some form of agriculture from Derby to Cooktown. Rice, sugar, cotton, peanuts, sorghum and some oil seed crops have been grown in successful commercial quantities, but producers have had heavy losses. Big projects, such as the damming of the Ord River for the purpose of agricultural production, are daring in their conception, especially in lands where living conditions are not easy, where transport and other costs are so high and where the choice of crops is extremely limited. Much has been proven in the field of research by the Commonwealth Scientific and Industrial Research Organization; but I know of no officer who would commit himself to the economics of agricultural production in the Northern Territory or in north-west Australia at this stage after long years of trials.

There is much concentration on rice at present, but cotton, with its enormous by-product value, may be the crop for our tropical north. America has 26,000,000 acres under cotton, producing 13,000,000 bales a year. In two States, California and Texas, it is grown without dependence on natural rainfall. The effect of cotton on a nation's diversified economy is enormouns. California treats 480,000 tons of cotton seed a year, and as a consequence, within reasonable distance of the cotton farms tens of thousands of cattle are "topped off" annually.

Such enormous feeding operations may ultimately exist in the Kimberleys and in north Australia. I agree that every endeavour should be made to use the irrigable areas in our far north, but the economics are not yet very clear. Ultimately the markets may be wholly based on overseas sales.

Mineral resources are known to be varied and considerable in our sparselands, and in them lies a great hope for towns to be developed even in remote places. Millions of acres still await careful prospecting. The enormous wealth from and in the massive deposits of silver-lead-zinc ores at Broken Hill, the vast deposits of copper ore at Mt. Isa, the asbestos at Wittenoom, which exceeds £300,000,000 in value, the gigantic bauxite deposits on Cape York Peninsula and at Gove in the Northern Territory, the iron ore of Yampi Sound and Mt. Goldsworthy, Roper River and other places, the thousands of tons of manganese of the desert areas, the radioactive materials so widely scattered, the mica at Harts Range, and the gold, beryl, nickel, tin, semi-precious and precious stones of the Pilbara and Coober Pedy are in our sparse areas.

In recent years large deposits of various minerals have been found in much-walked-over country, and in 1961 opportunities still await the prospector. The mineral possibilities are of the greatest, but real incentive must be given to both speculator and investor in mining enterprises. The climatic disadvantages, the isolation and the high installation and operational costs justify generous taxation concessions to residents and to all investors.

The mining industry makes a significant contribution to our overseas trade — £70 million and more in value annually. It gives us a stimulated internal industry. The demands of industry are so varied nowadays that no nation possesses all the minerals required for its own use.

Our mineral resources are superior to some of the highly industrialized European countries. No other comparable area in the world contains so many untested mineral prospects. Roads, airports, harbour facilities and other amenities are quickly inspired by a good mining operation. Artisans, shareholders and companies must all be assured of reward over and above that obtainable in more favourable regions. The resources are there, perhaps enough to pay our national debt. Let us go forward, therefore, through the medium of experts from the Government Bureau of Mineral Resources, by the use of scintillometer and aerial surveys, and giving full encouragement to experienced companies and skilled prospectors.

Of oil, it may be said that we have extensive beds of the right geological age, and it is surely unlikely that Australia will prove to be the only three million square miles of populated country on earth in which no oil is found in commercial quantities. At this very moment some oil is available at Exmouth Gulf at the turn of a stop-cock.

I now pass to a sentence or two in comment on the vast marine wealth in the waters adjacent to the shores of our sparselands. Whole nations with standards much different from ours could live from the seas abutting our shores. Whales pass up and down the coast in thousands, and pearl shell exists in large areas of our ocean beds. We know that the Japanese annually take shiploads of fish not far from our shores, and our coastal ships steam through miles of shoals of fish on every journey. Turtles abound over hundreds of miles of our coastline. We have allowed the Japanese to take thousands of tons of pearl shell from waters north of Australia in recent years. Australia gets very little from its northern marine wealth, but imports thousands of tons of fish from overseas.

In anticipating the possible spread of settlement what are the circumstances of climate affecting health? Much has been done in tropical health matters by Dr. Sir Raphael Cilento, and I am not unmindful of the valuable work done by such men as Dr. C. E. Cook and Dr. A. H. Humphry. The Flying Doctor Service, aerial medical services generally, the advent of refrigeration, better air services and the School of the Air have in recent years assisted in the mental outlook and the comfort factors which are so vital. One of the serious disabilities in living in our coastal tropical areas is the irritation caused by biting insects, notably sandflies and mosquitoes. A most important survey of environmental problems in tropical Australia was made in recent years by Dr. R. K. Macpherson of the National Institute of Medical Health, London. With deep humility I approach this subject, but with confidence do I quote this eminent man.

Dr. Macpherson stressed strongly the need for organized research directed towards the study of problems of tropical living. He regards as an urgent matter the identification of the factors which result in inefficiency, ill health, discomfort or discontent. Education for tropical living is most necessary. A difficult climate is no bar to successful living if the existence of the problems it creates is acknowledged and active measures are taken to solve them. In his comments on the effect of environment on health, he states that, excluding illnesses common to all areas of the world, the pattern of disease in our tropical north is malaria and other fevers, the bowel disorders, skin diseases and tropical debility. Of these, skin diseases and tropical debility are considered to be largely the product of environment. Dr. Macpherson considers that there is urgent need for research on the physiology of normal skin and the part played by heat and light in the causation of diseases of the skin. The problem of tropical neurasthenia is largely unsolved.

For several years, as Administrator of the Northern Territory, I worked closely and in collaboration with many men of high standing in the medical profession. To this great learned audience I would not presume to give advice, and humbly submit some views and thoughts which have been arrived at after much collaboration with such men.

In spite of the tropical climate and undeveloped state of the tropical north of Australia, endemic tropical diseases are few in number, and affect the European population very little. The native populations of the same latitudes in Africa and America are subject to many more diseases and to epidemics which have from time to time decimated the population. The Australian tropics are entirely free of such scourges as sleeping sickness, yellow fever, smallpox and malignant malaria. The tropical diseases which do occur are hookworm infestation, trachoma, yaws, leprosy, benign malaria, bacillary dysentery and ulcerating granuloma.

The health of the aborigines has been a matter of constant investigation and care by our medical men. Until the beginning of the century, the aborigines lived a tribal life, roaming only within their own tribal lands and having little or no contact with their neighbours. As a result they remained relatively free of transmissible disease, despite their primitive and insanitary way of life. Disease occurred only after the arrival of outsiders, who introduced infectious diseases and broke down the tribal barriers, and thus caused the spread of infection amongst the native population.

For many years there was a very real threat that the tables would be turned. With the encroachment of white settlement into remote areas, more and more contact between Europeans and aborigines occurred, and the aborigines threatened to hand back the diseases they had received in the past. However, since the spread of white settlement has been slow, health services have had time to exercise more and more control over the endemic diseases. This control, aided by modern advances in treatment, has considerably diminished the threat.

The greatest single risk in northern Australia above the seventeenth parallel is that from malignant malaria. Only a mild form of benign malaria at present exists in foci in and around Arnhem Land, especially along the course of the Roper River. Since 1955 less than 10 cases have been reported each year, but in 1955, after a long and heavy wet season, an estimated 150 cases occurred. Under similar weather circumstances, malignant malaria was

introduced in 1931. It spread along the Roper River into Katherine and Mataranka, and thence to Victoria River Downs and Wave Hill, entering Western Australian at the Ord River Crossing, and extending into the northern areas of the Kimberleys. Over 300 aborigines died. It is a constant fear of public health authorities that the disease will be reintroduced, for if it became established it would be a severe setback to the development of the north.

During the war, Army medical staff were so concerned at the possibility of the introduction of malignant malaria that troops who had recently been in malarious areas, such as New Guinea, were not permitted on the Australian mainland north of the seventeenth parallel until they had been out of malarious areas for six months. In the few cases of malaria that did occur among troops in the north the patients were immediately flown out of the area. The risk is still a real one, but it has been somewhat lessened by the production in recent years of more effective antimalarial drugs, by the speed at which aerial medical assistance is available and by the constant alertness to the risk by medical personnel trained in tropical diseases. I pay great tribute to them.

Hookworm occurs only in high-rainfall areas, and affects 80% of the native population of these areas. It will be eliminated only when the aboriginal learns elementary hygiene and ceases to foul the ground around his camp. It is most debilitating to young children, particularly when associated with poor nutrition. It is unlikely ever to be a serious problem to the European population.

Trachoma is very widespread among aborigines in both moist and dry areas, and is most severe in the latter. It is the cause of a good deal of blindness among central Australian aborigines. It is of a milder variety in the north. In the centre over 90% of the aborigines are infected; in the north the incidence varies from 50% to 80% in different localities. The disease is highly contagious and many Europeans become infected. A European usually seeks medical treatment early for inflamed eyes, and provided the treatment is early and thorough, the risk of serious consequence is much diminished. It will take repeated treatment over many years to eliminate the disease among aborigines.

Leprosy is endemic among the aborigines of northern Australia and attained a high incidence of over 5%. In Queensland it was introduced by Chinese and Kanakas, and in the Northern Territory and Western Australia by Chinese. The disease had become established in Queensland and the Northern Territory before 1900, but it first appeared in Western Australia near Roebourne in 1908. The disease spread rapidly through the most tropical areas, but Queensland took early vigorous action and the disease has now almost died out in that State. Control came more slowly in Western Australia and particularly in the Northern Territory, and the disease gained a firmer foothold. Leprosy is diminishing in these areas, but it will probably be another 20 years before it ceases to be a problem. The disease has not a high infectivity, but some 40 Europeans are known to have contracted leprosy in the Northern Territory in the past 70 years. As control progresses, this disease will become less and less a problem to white settlement, and it will not be a factor of importance in the development of the north by Europeans.

Ulcerating granuloma is a venereal disease almost entirely confined to coloured races. It has had only a small incidence among the aborigines and is dying out with treatment.

Epidemics of bacillary dysentery and other diarrhœal diseases will always be more common in warmer climates than in temperate ones, particularly among aborigines and pioneering outback communities of Europeans before proper sanitation is established. Dysentery and gastro-enteritis cause a higher number of deaths among children in the tropics than in temperate climates, but this is countered by greater freedom from severe epidemics of the common infectious diseases and chest complaints.

The health position therefore is bright, and perhaps the most spectacular change is that which has followed the widespread use of the aeroplane by the medical service. In all isolated areas, there is now constant touch with the outside world, and a sense of comfort and security is engendered by the knowledge that, in the event of illness or accident, skilled help is within radio reach.

Reference has been made to transport costs as one of the difficulties of setlement. The effects of transport costs on every phase of living and of industry in our outback are far-reaching, and seriously prejudicial in their effect. Costs of everything associated with living and family life are enormous, and the greatest factor in such costs is that of freight or transport. The transport needs of empty Australia are bound up with the existing and the potential patterns of land use, and with agriculture not yet reliably assessed, our development and anticipations must be based on the pastoral and mining industries. The provision of ports, railways, roads, stock routes and aerodromes is accepted as a Government responsibility. In this case the Commonwealth Government and four State Governments are involved, and it is a mater of great concern for all taxpayers.

There is talk at the moment of £20 million being spent on roads in far north Queensland to and through the Northern Territory, and about £7 million in north-west Australia. This is an excellent national move, even though belated, and requires an organized speeding-up with a continuous operation of road construction. Ports and railhead links with long distances of sealed roads in the north, centre and west of the continent are of the highest priority.

To get the best from our occupied areas and to avoid the annual colossal wastage in cattle, large sums are necessary to provide some penetrating railway extensions and long distances of sealed roads. Without some national scheme for transport facilities only a small percentage of the calves born in our remote areas will reach killing works as fat cattle. The magnitude of this national loss is appalling.

A remarkable increase in turn-off of stores and fat stock would result from the ability to use road or rail, as against the months of walking along stock routes. It is a matter for early determination how far it is practicable to meet the needs of remote places in transport requirements within national economic limits. It may be that defence aspects have a vital place in such determinations.

At present, materials for development, for homesteads and for daily use cost up to five times the initial cost in a capital city by the time they reach their remote destination. It is reasonable to expect that public investment in transport facilities will precede private investment in any productive activity. It must be made both possible and profitable for private enterprise to spend large sums in developing latent resources. A subsidy of a generous kind within the economic limits of the nation in the development of outback transport facilities would be a national investment. In the end it will repay the millions involved, and an annual commitment as meagre as 2% from the Commonwealth Budget would be realistic and within a decade transform our ways of production and of life in outlying parts.

Those who work and live there, and those who invest their money there deserve some real incentives.

Some comments on perhaps the greatest need of all I have left to the end of my address. That greatest need is to have generous taxation concessions given to residents and business enterprises in a very large area of sparsely occupied Australia. At the same time there is some fiddling with the problem in the granting of meagre zone allowances. When it is realized that the same payroll tax applies, that the same insidious sales tax operates, and that tax is paid on petrol on the same basis as in Perth or Sydney, some relief decision is long overdue. Freedom from taxation on all fuel used for air and road

transport, and for primary production in areas to be defined, is highly desirable. It is in the income tax field that proper action could give quick results.

All will agree that for living, for education of children, for development and business investments generally, some generous compensations of a substantial kind through income tax remissions would be realistic and immediately productive of results. A body known as the Northern Rehabilitation Committee has striven in vain for 14 years to influence governments to adopt a plan which appears to have all the ingredients of practicability. The plan is designed to assist the wage earner and his family, and to attract and keep in the north the skilled artisan and stockman. It is aimed at making remote areas so attractive that private capital, out of self-interest, would be anxious to seek investment there, and in spite of itself, develop and populate the area.

The proposal is that all territory north of the twenty-sixth parallel in Western Australia, the whole of the Northern Territory and an area of Queensland shall be declared a tax-free area for a period of 20 years for all wage and salary earners; that all business enterprises operating in the defined area, be they mining, pastoral, pearling, fishing, whaling, store-keeping or any other business, whether carried on by limited liability companies, partnerships or individual owners, whether resident or non-resident in the area defined, shall have 60% of their income from the area tax-free. The remaining 40% shall also be tax-free, provided it is invested within the defined area.

Such a proposal would offset many difficulties known to exist and would cost little more than £2 million a year—very little in £1300 million budget, and a stake with the prospect of far-reaching beneficial results. If such a scheme is successful in persuading people and capital to enter the north, producing more wealth and food, it would bring capital and labour together on a lasting basis with a prospect of high rewards to both nation and individual.

Surely Australians in a vast majority would be prepared to say to such a plan for the wide objective of peopling and developing our sparselands: "Go forth, make as much money as you like" in the hope that a government will cease being scared that somebody might make some money without paying tax.

We are obviously dealing with a problem country with an area that has resemblance to many sparsely populated parts of the world, but we are not dealing with a country without hope or resources.

Let us all assist somewhere, somehow, to improve the use and occupation of that part of our continent which, for national reasons, must be better used and more closely settled than now.

Our sparselands are capable of enormously increased production. It is a national matter of the most urgent kind. Commonwealth Government and State Governments might well combine to create an authoritative expert body with power to act and not merely to advise. Land laws and mining laws should be revised by the States to meet the situation, and all effects of taxation laws ought to be modified by the Commonwealth, even to the extent of amending the Constitution.

In addition to the taxation remissions suggested, the setting aside initially of a sum representing 2% of taxation collected by the Commonwealth Government would give £23 million annually, and if applied for 20 years, would mean a different Australia.

My humble endeavour has been to provoke thought on this subject in the earnest hope that we may inspire an enthusiasm in millions of Australians towards seeking a solution of this national problem.

GLUCOSE-6-PHOSPHATE DEHYDROGENASE ACTIVITY IN ANÆMIC PAPUANS.

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AND

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DURING an investigation into various types of anæmia occurring in Melanesians, it was noticed that several patients were suffering from an acute hæmolytic anæmia for which transfusion as an immediate treatment was often necessary. Subsequently these patients were given oral iron therapy and attained a satisfactory level of hæmoglobin quite rapidly and were not seen again. It was realized that this could be blackwater fever, as although this does not normally occur in adult native inhabitants of hyperendemic areas of falciparum malaria, it can occur if anti-malaria control measures are incomplete, particularly if suppressive drug treatment is used inefficiently (Mackie *et alii*, 1957). It is believed that because of the partial loss of naturally acquired immunity a sensitive state is created, leading to hæmolysis when reinfection with *Plasmodium falciparum* occurs (Adams and Macgraith, 1960).

However, several patients suffering from hæmolytic episodes in Port Moresby did not come into this category, and it was thought that possibly a deficiency of glucose-6-phosphate dehydrogenase might be the cause, hæmolysis being precipitated by drugs or an infection. Subsequently cells from patients with other types of anæmias were tested and it is the purpose of this article to report on the findings.

Patients Studied.

Twelve apparently healthy Melanesians were used as controls, and 45 patients were studied. Twelve were adults with acute hæmolytic anæmia of undetermined cause. The remaining 33 patients suffered from anæmia due to a variety of causes. Twenty-three were children and 10 were adults. Anæmia was due to thalassæmia major (four cases), thalassæmia minor (six cases), congenital hæmolytic anæmia of undetermined cause (10 cases), iron deficiency anæmia (10 cases), and anæmia secondary to severe tuberculous infection (three cases).

Methods.

Blood was collected into heparin, refrigerated and dispatched by air to Melbourne, usually not arriving until the following day. Occasionally examination was delayed longer than this, but the results quoted do not include specimens in which hæmolysis had occurred. Confirmatory examinations were made on some specimens collected into acid citrate dextrose (including Cases I and II) but the results did not differ significantly from those previously obtained. Glucose-6-phosphate dehydrogenase activity was measured by a modification of the method described by Zinkham *et alii* (1958), but the units were calculated as indicated by Hsia (1959). In this way the normal range is 150 to 210 units per 100 ml. of red cells for white Australians, agreeing well with the normal range quoted by Hsia (1959). It would appear that this was also close to the normal range for the twelve Melanesian controls. The enzyme appears to be stable for well over a week as long as the red cells remain intact (Parsons, personal observations).

Results.

The results are shown in Table I.

Reexamination of the case of thalassæmia major giving 470 units originally, during what appeared to be a hypoplastic episode, resulted in a new reading of 138 units. At this time the reticulocyte count was 0·1%. A further examination of the case of hæmolytic anæmia showing 315

units gave a reading of 212 units. At this time the patient's condition was much better and the reticulocyte count was only slightly increased.

Of the patients with iron-deficiency anæmias, three were pregnant and the remaining seven were children. Three of these had a second examination and in only one case was there a significant difference in the findings. This was the first case listed in the table, and on subsequent examination the reading was 260 units, when the patient's condition was clinically and hæmatologically greatly improved.

Of the patients with acute hæmolytic anæmia, only the last was reexamined. Testing showed 8 and 11 units per 100 ml. of enzyme activity when repeat examinations were made. The three tuberculous patients were all children with moderate anæmia. It was not possible to locate again the last child listed in the table so that this low result remains unconfirmed.

TABLE I.

Glucose-6-Phosphate Dehydrogenase Activity in Anæmic Papuans.[1]

Condition.	Number of Patients.	Glucose-6-Phosphate Dehydrogenase Activity. (Units per 100 ml. Red Cells.)
Thalassæmia major ..	4	700, 470 (138),[2] 518, 56 (46), (36)
Thalassæmia minor ..	6	382, 285, 258, 258 (229), 192, 170
Acute hæmolytic anæmia of undetermined cause (adults).	12	730, 610, 440, 415, 390, 380, 380, 340, 338, 270, 187, 0 (8) (11)
Congenital hæmolytic anæmia of undetermined cause.	10	645, 430, 316, 315 (212), 311, 298, 269, 261, 248, 193
Iron-deficiency anæmia	10	490 (260), 485, 402, 370, 350, 325, 298, 264, 215, 204
Anæmia due to tuberculosis.	3	286, 234, 66
Healthy controls ..	12	147, 175, 183, 188, 184, 146, 178, 150, 161, 220, 227, 200

[1] The normal range is 150–210 units per 100 ml. of red cells.

[2] The figures in parentheses indicate repeat examinations.

From the above reports it will be seen that only three patients gave evidence of low enzyme activity. One of these, a child with what appeared to be a mild hæmolytic anæmia secondary to generalized tuberculous adenitis, could not be reexamined, and it is not proposed to discuss her further. Of the remaining two, one child is suffering from thalassæmia major and has been more fully described elsewhere (Ryan, 1961), so that only a brief description will be given here. The third patient, an adult who showed no or very low enzyme activity, will be fully described.

Reports of Cases.

Glucose-6-Phosphate Dehydrogenase Deficiency.

CASE I.—A male Papuan, aged 5 years, a native of Kerema, was admitted to the Port Moresby General Hospital on numerous occasions over a period of three years for transfusion, because of a severe hæmolytic anæmia. He was finally diagnosed as suffering from thalassæmia major, after splenectomy on May 23, 1960. Since operation he has been reasonably well, but his hæmoglobin level remains at about 7 grammes per 100 ml. A transfusion of one pint of blood was given on September 21, 1960, when his hæmoglobin level was 4·8 grammes per 100 ml., but no further treatment has been required. Glucose-6-phosphate dehydrogenase level estimations were done on June 21, 1960, and September 2, 1960, and were reported as 56 and 46 units respectively. A further estimation on January 6, 1961, gave the figure 36 units. There has been no evidence of drug-induced hæmolysis at any time, or sensitivity to *Vicia faba*, and it is not considered legitimate to try the effect of "Primaquine", because of his thalassæmic condition.

CASE II.—A male Papuan, aged 36 years, a native of Rigo, was admitted to the Port Moresby General Hospital on June 12, 1960. He gave a history of "fever" for the previous week, with occasional rigors, and said that he had passed dark urine. On the day before admission, he had vomited several times, and had developed pain in the left hypochondrium. On examination he was noticed to be very pale, to have slight generalized œdema and to be icteric. The heart and lungs appeared normal, but the spleen was enlarged so as to be palpable at the umbilicus, and the liver was palpable two fingers' breadth below the right costal margin. The hæmoglobin level was 3·9 grammes per 100 ml., the packed-cell volume was 15%, the mean corpuscular hæmoglobin concentration was 26% and a white-cell count yielded a total of 9700, with a normal distribution. Examination of a stained smear showed hypochromia, polychromasia and aniscocytosis, but there was little poikilocytosis. Few normoblasts were seen, and the reticulocyte count was 3·9%. No malaria parasites were seen. The serum bilirubin level was 1·5 mg. per 100 ml. and the urine contained excess urobilinogen. Examination of the stools for occult blood gave a positive result on two occasions, but no cysts or ova were seen. The Coombs test gave a negative result. Liver function test results were normal. Radiological examination of the skeleton revealed no abnormalities, and this was so also for the stomach and intestine. On June 24, 1960, an alkali-denaturation test revealed only 1% of undenatured hæmoglobin, and hæmoglobin electrophoresis showed no abnormal hæmoglobin to be present. Liver function tests also gave normal results. At the same time examination of the sternal marrow disclosed advanced erythroid hyperplasia. The treatment consisted of transfusion and several injections of "Imferon". Four days after the latter was commenced, the reticulocyte count was 11·6%. On September 1, 1960, glucose-6-phosphate dehydrogenase activity was reported as nil. That this was not due to inhibitors was shown by the addition of enzyme from a case of thalassæmia (518 units per 100 ml.), which brought about an excellent recovery. The Coombs test again gave a negative result. Further questioning at this time elicited the information that the patient had been taking "Camoquin" as suppressive therapy in a dosage of two tablets weekly for some months. He also stated that his brother had become yellow and œdematous, and had died suddenly about one year previously. He denied that he or his brother had ever eaten any type of beans. A routine course of "Camoquin" was given six weeks after his admission to hospital, but there were no ill effects, even though this was repeated twice in the following two weeks. The results of hæmatological examination at this time were essentially normal and the patient was discharged. He was readmitted for further investigation on October 1, 1960, and 30 mg. of "Primaquine" were given daily for 10 days, but there was no evidence of hæmolysis, the hæmoglobin level remaining constant at 12·2 grammes per 100 ml. and the serum bilirubin level normal. Reticulocyte counts on several occasions also gave normal results. A glucose-6-phosphate dehydrogenase estimation again on this occasion gave evidence of very low (8 units) activity. This was again confirmed subsequently on February 8, 1960. On this last date the hæmoglobin level was 15·4 grammes per 100 ml. and the glucose-6-phosphate dehydrogenase activity was 11 units per 100 ml.

Greatly Increased Glucose-6-Phosphate Dehydrogenase Activity.

CASE III.—A Papuan male, aged three years, a native of the Oro Bay district, was admitted to hospital on September 27, 1960, with a history of anæmia from the age of three to four months. He had received numerous transfusions. Clinically his condition was suggestive of thalassæmia major, with enormous hepatosplenomegaly, bossing of the skull and mongoloid facies. Hæmatological examination disclosed a hæmoglobin level of 4 grammes per 100 ml. a red-cell count of 1,960,000 cells per cubic millimetre, a packed-cell volume of 13%, a mean corpuscular hæmoglobin concentration of 31%, a mean corpuscular volume of 65 c.μ, 2200 nucleated red cells per cubic millimetre and a reticulocyte count of 16%. A white-cell count totalled 9900 cells per cubic millimetre, with a normal distribution. Examination of the peripheral blood showed anisocytosis, poikilocytosis, polychromasia and target cells. An alkali-denaturation test showed the presence of 46% of undenatured hæmoglobin, and hæmoglobin electrophoresis showed a major component with the mobility of hæmoglobin F. The Coombs test gave a negative result. At the time of admission to hospital the patient had received no transfusion for four months. A glucose-6-phosphate dehydrogenase activity estimation at this time gave a figure of 700 units. This specimen was in good condition on arrival.

CASE IV.—A Papuan female from the Central District, aged about 22 years, was admitted to hospital on August 20, 1960. She said that for the previous two weeks she had suffered from joint pains and had increasing lassitude, fever with occasional rigors and headache, and had passed red urine for two days shortly after becoming ill. She stated that she had been taking suppressive doses of "Camoquin" regularly for some months. On examination she had marked pallor

of the mucous membranes and her scleræ were icteric, her spleen was enlarged and palpable four fingers' breadth below the left costal margin, and she appeared to be about 26 weeks pregnant. Hæmatological examination showed a hæmoglobin level of 3·2 grammes per 100 ml., a red-cell count of 1,100,000 cells per cubic millimetre, a mean corpuscular hæmoglobin concentration of 29%, a packed-cell volume of 11% and a white-cell count of 3566 cells per cubic millimetre, with a normal distribution. The halometer reading was 8·1 units. Normoblasts were present in the peripheral blood (1104 per cubic millimetre). The erythrocytes showed anisocytosis and polychromasia, and 35·7% were reticulocytes. Urobilinogen was present in excess in the urine. Examination of the bone marrow showed intensely hyperplastic erythropoiesis, which was macro-normoblastic in type. Granulopoiesis appeared normal. The Coombs test gave a negative result. Radiological examination of the skull, hands and feet was normal and an alkali-denaturation test and hæmoglobin electrophoresis both gave normal results. Assay of the urine for formiminoglutamic acid gave a negative result. There was no response to a régime of iron, folic acid and cortisone over a period of 16 days. The patient was then given a blood transfusion and her hæmoglobin level continued to rise slowly after this, and was 10·5 grammes per 100 ml. two months after her delivery, which occurred at thirty-two weeks' gestation. A glucose-6-phosphate dehydrogenase estimation performed one week after her admission to hospital yielded the figure of 645 units per 100 ml. of red blood cells. Repeated examination of the peripheral blood failed to show malaria parasites.

Discussion.

Since the reports by Cordes (1926) and Manson-Bahr (1927) of hæmolytic anæmia following the ingestion of plasmochin, many other observers have reported hæmolysis following the ingestion of "Primaquine". It was noticed during World War II that "Primaquine" sensitivity was much commoner amongst African, Indian and Burmese troops than amongst Europeans. In fact this sensitivity is rare amongst the latter. Dern, Beutler *et alii* (1954) reported that about 10% of American Negroes developed hæmolysis after ingestion of 30 mg. of "Primaquine" daily. They demonstrated that this susceptibility to "Primaquine" lay in the erythrocytes themselves, and was associated with a low reduced glutathione content of the cell, approximately only two-thirds that of normal persons. They also showed that there was glutathione instability when these cells were exposed to acetylphenylhydrazine. Glutathione comprises the major component of the non-protein sulphydryl compounds of the red cells, and has widespread biological importance. It is essential for cell division, shows a protective action against certain poisons and X-ray irradiation and participates in several enzymatic reactions (Szeinberg *et alii*, 1957). All cases in the series quoted by the latter authors, which were associated with a past history of favism, or hæmolytic anæmia due to sulpha drugs and para-amino salicyclic acid, were distinguished by a low concentration of glutathione in their erythrocytes. Beutler *et alii* (1954) also showed that sensitive cells formed Heinz bodies more readily than normal cells, when incubated with acetylphenylhydrazine. They demonstrated that the formation of these bodies was the initial step in the hæmolytic process and that they disappeared as the hæmolytic process continued, suggesting that cells containing them were selectively destroyed. They showed that these cells were also sensitive to drugs such as sulphanilamide and "Diasone" and Giles and Ikeme have demonstrated glucose-6-phosphate dehydrogenase deficiency in Nigerians sensitive to phenacetin and naphthalene (1960), as have several other workers.

Carson *et alii* (1956) demonstrated a deficiency of the enzyme glucose-6-phosphate dehydrogenase in "Primaquine"-sensitive erythrocytes, and it is thought that this is responsible for the instability of the glutathione in these cells. In fact the results of glutathione stability tests and glucose-6-phosphate dehydrogenase estimations coincide well, but occasional divergences have been reported (Szeinberg, Sheba and Adam, 1958). A comparatively high incidence of glucose-6-phosphate dehydrogenase deficiency has been found amongst relatives of susceptible persons, suggesting that a genetic defect is the cause of the lowered enzyme content. It is well known that genes may express themselves in terms of qualitative alterations of macromolecules such as human hæmoglobins (Ingram, 1957) and blood group substances (Kabat, 1956). However, it seems more likely that in the case of "Primaquine" sensitivity we have to consider the possibility of a gene, which manifests itself through decreasing the rate of synthesis, or increasing the rate of inactivation of this enzyme (Kirkman, 1959). It appears that the gene is sex-linked, as men can be divided into reactors and non-reactors, whereas women in the families of affected persons cannot be so classified (Beutler, 1959). That it is incompletely dominant has been shown by Childs *et alii* and by Szeinberg *et alii* (1958). Larizza *et alii* (1958) and Zinkham *et alii* (1958) demonstrated the same type of sensitivity in favism, although in these cases the hæmolytic process may be much more severe (McPhee, 1956). Szeinberg *et alii* (1958*b*) have also demonstrated the same type of defect in non-Ashkenazic Jews sensitive to sulpha drugs.

Two types of glucose-6-phosphate dehydrogenase deficiency have been found—the negroid type with about 10% activity, as shown by Case I and perhaps the other case (not described) with 66 units per 100 ml., and the Caucasian type with 0–1% activity, as shown by Case II (Marks and Gross, 1959). It is unusual to find the two types existing in the one population, but it can be stated that the patient described in Case II, as far as facial appearance and colour are concerned, like many Papuans, could pass as a Caucasian, whereas the first two patients described are definitely negroid in appearance. It is difficult to understand why the patient in Case II did not develop hæmolysis when given "Primaquine" for 10 days. The dosage of 30 mg. daily is the standard dose prescribed for testing "Primaquine" sensitivity, and hæmolysis usually begins two to three days after the drug has been given and ceases in about a week, when regeneration begins (Beutler, 1959). A possible explanation is that he had not fully recovered from his hæmolytic episode, as a refractory period of several weeks has been reported normally in these cases following a hæmolytic attack. This does seem unlikely as the "Primaquine" test was not carried out until three months after the attack of hæmolysis. However, it will be noted that at this time the patient's hæmoglobin level was 12·8 grammes per 100 ml., whereas it was subsequently 15·2 grammes per 100 ml. Obviously the possibility of his still being in a refractory phase cannot be excluded. However, it should be remembered that while people whose erythrocytes are susceptible to hæmolysis by "Primaquine" or *Vicia faba* are glucose-6-phosphate dehydrogenase deficient, it remains to be proved that the reverse is true in all cases. In fact, people with a low glutathione content, amongst relatives of patients with favism, have claimed to have eaten fava beans without harm to themselves (Szeinberg *et alii*, 1957). McPhee (1956) also quotes several examples of people with favism, who had previously eaten the beans on several occasions without an attack of hæmolysis. However, special factors probably operate in favism. It is known that several hundreds of Melanesians from the New Guinea highlands have been treated with "Primaquine" without apparently producing hæmolysis, although it is realized that slight attacks might not be reported or observed (Peters, 1961). However, this does suggest that drug sensitivity is not very common in this particular group. Johnson and Marks (1958) have reported that glucose-6-phosphate dehydrogenase activity is higher in young than in old cells, and that the difference is particularly large in "Primaquine"-sensitive cells. For this reason it might be expected that all cases of thalassæmia would show high glucose-6-phosphate dehydrogenase content, as shown in Case III, and in the other two cases not described. Also it would be expected that if hæmolysis did occur during transportation of blood, the older cells would be destroyed first, and that high values would be reported. This is a possible explanation of the high values recorded in three of the thalassæmia cases examined, but there was no hæmolysis obvious in the specimens at the time of examination. It is much more likely that the results obtained were due to the presence of many young cells

and reticulocytes. It is also possible that the increased numbers of cells present per unit of volume in the microcytic anæmias might lead to higher values than normal. This is particularly likely to occur in transported blood, as cell shrinkage is known to occur during storage. However, in the method used here hæmoglobin was used as the primary reference to overcome this latter possibility. Beutler *et alii* (1954) considered that older cells were selectively destroyed, and that this was, in fact, the cause of the self-limiting course of hæmolysis due to "Primaquine" sensitivity. For this reason it is probably unusual to find glucose-6-phosphate dehydrogenase activity reduced in the patient of Case I, who is suffering from thalassæmia major. However, Kidson (1960) states that he has seen this combination in several American Negroes, and Newton and Bass (1958) reported glutathione instability and a total absence of glucose-6-phosphate dehydrogenase in the blood of three Caucasian children suffering from congenital non-spherocytic hæmolytic anæmia. Naylor *et alii* (1959) also reported glucose-6-phosphate dehydrogenase deficiency and glutathione instability in eight persons with homozygous sickle-cell disease, in three persons with sickle-cell hæmoglobin C disease, and in two with sickle-cell trait. In all instances the two defects segregated independently of each other. Family studies in the two cases with enzyme deficiency are incomplete, but so far no sibs seem to be affected.

As a practical point, it is worth noting that "Primaquine"-sensitive cells may hæmolyse more readily under the stress of infection. Szeinberg *et alii* (1957) reported that gross blood destruction is encountered in certain population groups susceptible to favism and drug sensitivity following some infectious diseases — for example, typhoid fever. They also reported that the incidence of hæmolytic anæmia following influenza is much higher in Sephardic Jews, who have a high incidence of this defect, than in Ashkenazic Jews, who have not. Kidson (1960) also states that hæmolysis in "Primaquine"-sensitive people may be precipitated by infectious hepatitis. It thus seems possible that the association of pneumonia with jaundice, described by Gelfand (1957) as frequently occurring in African Negroes, may not always be due to liver breakdown, but may also be associated with enzyme deficiency. A similar association of pneumonia and jaundice is also relatively common in New Guinea (Campbell, 1960), so that it would probably be worth while investigating the association, if any, of glucose-6-phosphate dehydrogenase deficiency and infections in this country.

It would seem possible that the hæmolytic episodes described in Cases II and IV may have been blackwater fever, as both patients had been taking suppressive doses of "Camoquin", and it is commonly impossible to demonstrate parasites in the blood at the time of the attack (Mackie, Hunter and Worth, 1957).

Summary.

A study of glucose-6-phosphate dehydrogenase activity in Papuans suffering from anæmia of various types is described. It is suggested that the acute hæmolytic episodes in these people may occasionally be due to glucose-6-phosphate dehydrogenase deficiency, but that this is not the usual cause. Two cases of this deficiency are described and possibly a third. Other cases in which greatly increased enzyme activity was present are described, and an explanation of these results is suggested. It is tentatively proposed that some cases of jaundice associated with infections in Papuans may be due to glucose-6-phosphate dehydrogenase deficiency, but no evidence of drug sensitivity has been produced at this stage.

It would seem unwise to give suppressive anti-malaria treatment to immune persons because of the danger of inducing blackwater fever, and especially unwise to use "Primaquine".

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DEFICIENCY OF GLUCOSE-6-PHOSPHATE DEHYDROGENASE: SOME ASPECTS OF THE TRAIT IN PEOPLE OF PAPUA-NEW GUINEA.

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INVESTIGATIONS in several laboratories on erythrocyte metabolic defects have clarified some problems connected with certain drug-induced hæmolytic anæmias. They have also proved to be challenging from the viewpoint of human biochemical genetics.

This work began with the implication of pamaquine (Earle *et alii*, 1948) and primaquine (Hockwald *et alii*, 1952) in the ætiology of hæmolytic anæmia in American Negro troops undergoing treatment for malaria. Many drugs and the fava bean have since been shown to have a similar effect (reviewed by de Gruchy, 1960). The extensive studies carried out to elucidate the underlying mechanism have been reviewed recently by Marks and Gross (1959*a*) and by Beutler (1959).

The self-limited nature of the hæmolytic process in primaquine-induced anæmia was first observed by Dern and his associates (1954*a*), who later (Dern *et alii*, 1954*b*) suggested that primaquine sensitivity was the result of an intrinsic abnormality of the erythrocytes in affected subjects. Beutler and associates reported in 1955 that the red cells of affected individuals were deficient in reduced glutathione, while Carson and coworkers (1956) found that these cells has a considerably diminished activity of glucose-6-phosphate dehydrogenase.

Utilization of glucose by the erythrocyte involves phosphorylation to glucose-6-phosphate and subsequent degradation via the reactions of anaerobic glycolysis or the pentose phosphate pathway (Marks, 1956; Horecker and Hiatt, 1958). The sequence of events in the latter pathway is illustrated in Figure I. In the first oxidative step the conversion of glucose-6-phosphate (G6P) to 6-phosphogluconate is catalysed by the enzyme glucose-6-phosphate dehydrogenase (G6PD), and results in the generation of reduced triphosphopyridine nucleotide (TPNH). This is the only known mechanism by which mature erythrocytes produce TPNH (Brin and Yonemoto, 1958), which is required as a cofactor for glutathione reductase in the conversion of oxidized glutathione (GSSG) to the reduced form (GSH).

Thus a deficiency of G6PD leads to a secondary decrease in TPNH formation, and a consequent limitation of GSH production, with apparent instability of reduced glutathione. There is a compensatory increase in glutathione reductase level (Alving, 1958).

Observations by several investigators suggest that the trait — deficiency of G6PD — in inherited as a sex-linked gene of intermediate dominance (Gross *et alii*, 1958; Szeinberg and Sheba, 1958; Childs *et alii*, 1958). Two groups of persons with reduced erythrocyte G6PD are evident: those with low levels, mostly males, and those with intermediate levels, who are females (Marks and Gross, 1959*a*). The suggestion has been proffered that the low and intermediate levels represent respectively the homozygous and heterozygous states (Childs *et alii*, 1958). However, Marks and Gross (1959*a*) have observed in one family a mother with a very low G6PD level who gave birth to four normal sons. These workers suggested that the phenotypic expression may be modified in the female or that the trait may be heterogeneous.

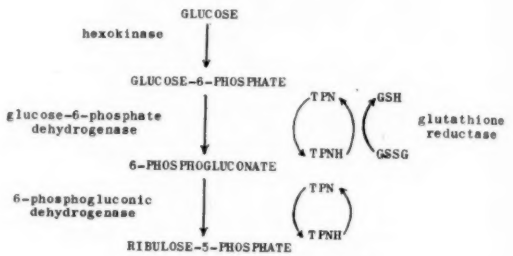

FIGURE I.
Reactions of the pentose phosphate pathway.

The distribution of the trait is scattered. First shown in American Negroes, it was later reported in Sephardic Jews (Szeinberg and Sheba, 1958), in Italians, primarily of Sicilian and Sardinian descent (Sansone and Segni, 1956), in subjects of Greek origin (Gross *et alii*, 1958), in Africa (Gillies *et alii*, 1960; Allison, 1960) and in Indian and Chinese people in Singapore (Vella, 1959).

Extensive studies have revealed differences between the enzyme activity in affected Caucasians and Negroes (Marks and Gross, 1959*b*). Thus, deficient Caucasian males, when compared to Negro males, were shown to have a significantly lower G6PD activity in the whole erythrocyte population; in affected Negroes the activity of the enzyme in the young red cells, though not approaching control levels, was higher than in old cells. In Caucasian males, whose erythrocyte population had little or no detectable G6PD activity, these workers showed that there were relatively small differences in the amount of enzyme detectable in young, as compared with old, red cells. In addition, affected Caucasians have low enzyme levels in leucocytes and platelets (Ramot *et alii*, 1959). From this work, Marks and Gross (1959*b*) suggested possible genetic interpretations to be: (i) different mutations occur in the same region of the X chromosome concerned with the formation of G6PD; (ii) more than one gene may affect the level of G6PD activity, and a second mutation in affected Negroes may result in partial restoration of enzyme activity; (iii) two or more distinctly different proteins may be produced which catalyse the same reaction.

The present work was undertaken in an attempt to find additional populations exhibiting the trait, which may help to elucidate the mechanisms governing genetic control of the synthesis of G6PD. Apart from this genetic significance, the finding of the trait in Papua-New Guinea is of clinical interest and of possible importance in anthropological studies. A preliminary report of portion of this work has appeared in note form (Kidson, 1961).

[1] This work was carried out during the tenure of a grant from the Anti-Cancer Council of Victoria.

ILLUSTRATION TO THE ARTICLE BY J. K. DAWBORN.

FIGURE II.
Histological section: lymphosarcomatous infiltration through the muscle of the small bowel (× 26).

ILLUSTRATION TO THE ARTICLE BY R. D. MACBETH.

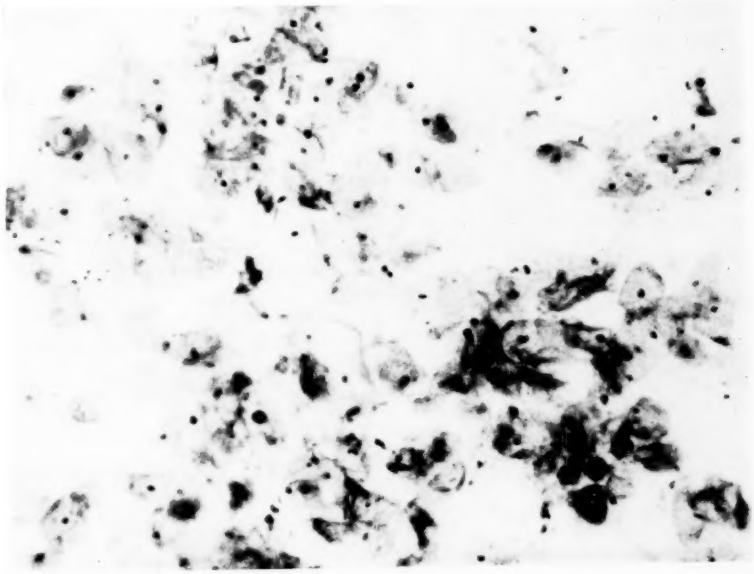

FIGURE V.
Typical field of epithelial cells in film preparation of liquor amnii. Very few of these nuclei are "readable".

ILLUSTRATIONS TO THE ARTICLE BY N. S. BUCKMASTER.

FIGURE I.
Bilateral common iliac artery emboli. The measurement is in centimetres.

FIGURE II.
Section of the lung growth showing oat-cell carcinoma. × 100.

FIGURE III.
Section of one embolus showing tumour tissue in organized thrombus. × 100.

Materials and Methods.

Samples of whole blood were collected in vacuum venules and transported by air from New Guinea to Melbourne. In most cases clots were employed, but in some instances blood samples were heparinized. All samples were refrigerated at approximately 4° C. between the times of collection and transhipment, but some exposure to air temperatures occurred during transportation.

Clotted and heparinized samples from normal subjects collected in Melbourne were stored both at 4° C and 25° C. for two to three weeks, and used as controls to determine the effects of storage and temperature. Glucose-6-phosphate dehydrogenase activity was estimated by a modification of the method of Motulsky and Campbell (1961). This method depends upon the colour change which occurs in the dye, brilliant cresyl blue, as it passes from the oxidized to the reduced form. Hydrogen ions released from glucose-6-phosphate by G6PD are accepted first by TPN and then transferred to the dye. Thus, the dye colour change is an index of the activity of the enzyme.

In the case of clotted samples the serum was removed, the cells washed from the clots and suspended in normal saline. The volume of saline was adjusted to give a standard hæmoglobin value. All samples showing hæmolysis were rejected. 0·02 ml. of blood or cell suspension was added to 1·0 ml. of distilled water in tubes of 10 mm. diameter. To this were added, with mixing, 0·2 ml. of a 0·74 M tris-(hydroxy-methyl)-amino-methane[2] buffer at pH 7·5, 0·1 ml. of a 0·05 M sodum glucose-6-phosphate[3] solution, 0·1 ml. of a 0·2% TPN[4] solution and 0·25 ml. of a solution of 1×10^{-3} M brilliant cresyl blue.[5] To prevent reoxidation of the dye by the air the mixture was topped with approximately 1 ml. of mineral oil and the tubes were incubated in a water bath at 37° C. The end-point was indicated by a colour change from blue to pink, observed against a constant white light source. This end-point could be estimated with an accuracy of ± 5 minutes, a small percentage error relative to the reaction times observed. For convenience reaction times have been graphed as rates of reaction in minutes^{-1} (Figures II and III).

Spectrophotometric assays were performed after the method of Kornberg and Horecker (1955) on some of the heparinized samples as a check on the screening method. The two methods gave comparable results.

Results.

Normal Values for G6PD.

The normal samples gave an end-point range of 50 to 100 minutes. End-points greater than 200 minutes were considered to represent deficiency of the enzyme. The majority of abnormal samples took 300 to 400 minutes for the colour change, but a few did not change until about 15 hours. There was a distinct gap in the range between the normal and abnormal time values.

G6PD Activity in Stored Blood.

The effects of time and temperature on the G6PD activity of erythrocytes in stored clotted blood are shown in Figure II. Both samples were from the same subject. It may be seen that in the sample stored at 4° C. there was no significant deviation from the initial value of enzyme activity even after three weeks. However, in the sample stored at 25° C. there was a moderately rapid rate of deterioration in the first few days, followed by a slow fall in activity over the succeeding weeks. This initial rapid decrease presumably represents hæmolysis of the older cells followed by denaturation of the enzyme, while the slower subsequent decrease in activity presumably means that there is then a more steady rate of hæmolysis of the younger cells, as indicated by the work of Marks, Johnson and Hirschberg (1958) (Figure II).

Figure III shows comparable results of enzyme activity in heparinized blood. The effects of storage were similar, with little change in the activity of the blood at 4° C. The blood at 25° C. showed a lag period before the activity began to diminish. This period was variable and in some cases absent.

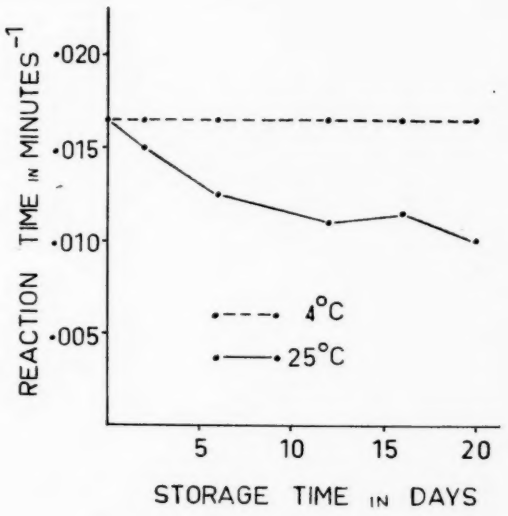

FIGURE II.
Effect of storage on glucose-6-phosphate dehydrogenase activity in clotted blood.

These results are supported by Marks *et alii* (1958), who showed that red cells stored at 4° C. in suitable media showed no change in the levels of G6PD activity over seven months.

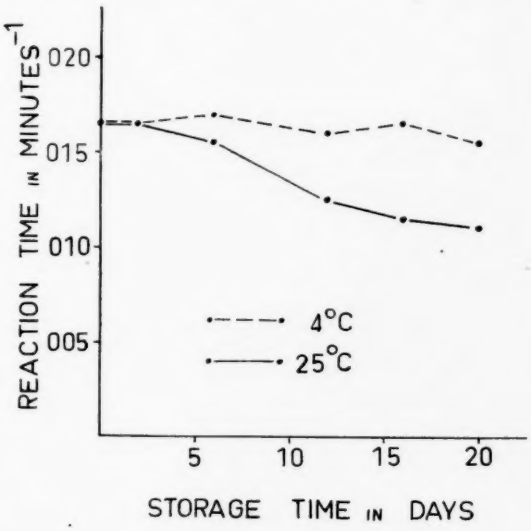

FIGURE III.
Effect of storage on glucose-6-phosphate dehydrogenase activity in heparinized blood.

Even after three weeks the levels of enzyme activity were not as low as the levels in fresh blood samples from persons with G6PD deficiency. Hence adequate reliance could be placed on enzyme estimations performed on blood samples under the conditions of transport and storage in this survey.

[2] Obtained from British Drug Houses Ltd., England.
[3] Obtained from Nutritional Biochemicals Corporation, Cleveland, Ohio, U.S.A.
[4] Obtained from Sigma Chemical Company, U.S.A.
[5] Obtained from G. T. Gurr Ltd., London, England.

G6PD Activity in New Britain.

The results of enzyme estimations on blood samples from 155 persons inhabiting the Gazelle Peninsula, New Britain, are given in Table I. The finding of 16 persons with deficiency of G6PD in their erythrocytes out of 155 subjects tested indicates that in this area there is a high incidence of the trait. Several females in this group had enzyme levels in an intermediate range, possibly suggesting a heterozygous situation, but the screening method is not sufficiently accurate to enable a precise assessment of such cases.

TABLE I.

Glucose-6-phosphate Dehydrogenase Activity in Blood Samples from the Gazelle Peninsula, New Britain.

Sex.	Number Examined.	Number Deficient.
Male	94	16
Female	61	0
Total	155	16

G6PD Activity in the Eastern Highlands, New Guinea.

The values for 534 subjects in the Eastern Highlands area of New Guinea are listed in Table II. These samples were collected mainly around Kainantu. Three cases of enzyme deficiency were detected in this group, giving a low incidence. The possibility of extraneous mixing in this group is small, but it is feasible that admixture has occurred between coastal and highland peoples.

TABLE II.

Glucose-6-phosphate Dehydrogenase Activity in Blood Samples from the Eastern Highlands, New Guinea.

Sex.	Number Examined.	Number Deficient.
Male	383	3
Female	151	0
Total	534	3

G6PD Activity in Coastal Areas of New Guinea.

The results on a total of 130 samples from three coastal areas, two in Papua-New Guinea and one in West New Guinea, are shown in Table III. In these small groups there was an incidence of 1 in 39 in Port Moresby, 2 in 69 in Wewak, and 3 in 22 in West New Guinea. Taken together, the six cases from coastal areas out of a total of 130 tested suggest a higher incidence than that in the highlands, but a lower incidence than that in New Britain. However, larger samples will be necessary to determine fully the significance of these differences.

TABLE III.

Glucose-6-phosphate Dehydrogenase Activity in Blood Samples from Coastal Areas of Papua-New Guinea and West New Guinea.[1]

Area.	Number Examined.	Number Deficient.
Wewak	69	2
Port Moresby ..	39	1
West New Guinea (Asmat)	22	3

[1] The sex was not recorded for all samples in this group, but all deficient samples were from males.

Discussion.

From these results it is evident that the inherited defect of erythrocyte metabolism, deficiency of the enzyme glucose-6-phosphate dehydrogenase, is present in at least several areas of Papua-New Guinea, including the island of New Britain, and in West New Guinea. Thus another distinct population has been shown to possess the mutant gene which has been demonstrated previously in Negroes in Africa and the United States of America, and in populations in the Mediterranean area.

The population studied here is isolated from these groups which have been investigated extensively. Nearer geographically to New Guinea the trait has been found in Indian and Chinese persons in Singapore, but not in Malays, thus far (Vella, 1959). Earlier, favism, the hæmolytic state in persons with this deficiency induced by eating fava beans, had been reported by several workers in China (Du, 1952; Wang and Wang, 1956; Bernard, 1959). Although the populations studied in New Guinea are considered to be fully indigenous, the likelihood of Chinese admixture is remotely possible. However, it is more likely that the trait represents a mutation arising from within the indigenous community.

The Mediterranean and Negro groups in which G6PD deficiency exists also exhibit the sickle-cell trait. However, thus far sickle-cell hæmoglobin has not been demonstrated in New Guinea or New Britain (Swindler, 1955; Walsh and Cotter, 1955). It is apparent therefore that these mutants need not occur concomitantly in the same population, a factor to be considered in studies of population migration in this area. However, thalassæmia has been reported in Papua (Ryan, 1961).

A marked difference in incidence is observed in the particular areas which have been screened for the defect. Especially noticeable is the difference between the Gazelle Peninsula in New Britain (high) and the Eastern Highlands (low). The figures given for the coastal areas of New Guinea may be less reliable because of the small numbers surveyed, but are also suggestive of a higher incidence in low-lying coastal, as opposed to highland, areas.

Motulsky *et alii* (1959, 1961), from a survey of mountainous and coastal groups in the Congo Republic, found that the incidence of the trait was low in highland areas and high in coastal areas, corresponding to the distribution of malaria. It is their contention that there may be a selective advantage in the development of the trait. They argue that since reduced glutathione is diminished and easily depleted in enzyme-deficient cells, and since malarial organisms require GSH for growth, enzyme deficient subjects may be more resistant to malaria.

The Gazelle Peninsula in New Britain is low-lying and highly malarious (Black, 1960). S. H. Christian surveyed the Eastern Highlands area in 1949-50, found no splenic enlargement or parasites in the Kainantu subdistrict (5000 feet) in 1949, but found malaria south-east of Kainantu (2950-4500 feet) in 1950. Some of the tribes in the Kainantu area trade down the valleys and come back with malaria from these excursions to lower altitudes (Schofield, 1960). However, while there has probably been some patchy spread in the last 10 years, the Kainantu area in terms of genetic history can be considered as low- or non-malarious. The coastal areas of New Guinea are of course highly malarious.

The finding in this present survey of a high incidence of the enzyme defect in coastal, high-malarial areas and of a very low incidence in the highland areas of New Guinea supports Motulsky's theory of the possible implication of malaria in selection in favour of the enzyme-deficient mutant.

No cases of primaquine sensitivity have been recorded in Papua-New Guinea, but Schofield (1960) suggests that this is not a very important observation, because primaquine is given to highland peoples only when they return from the coast to their own malaria-free or comparatively non-malarious home areas. The drug is not used in treating peoples on the coast because of the high rate of reinfection. So far as is known (Schofield, 1960) no native food has been associated with hæmolytic anæmia.

Stress has been laid on the fact that the screening test employed here cannot be used for very accurate quantitation. However, attention is drawn to the finding that while the enzyme in most affected subjects produced a colour change in the dye at about 300 minutes, several samples

gave no change until 15 hours. The finding of quantitative differences between Negro mutants, who show 10%-20% enzyme activity, and Caucasian mutants, who show 0-1% enzyme activity, is considered (Marks and Gross, 1959*b*) to represent the involvement of multiple genetically determined factors in the expression of this enzyme. Further studies are in progress in this laboratory to determine the possibility that two or more different mutants may exist in the New Guinea area. No definite conclusions can yet be drawn. Investigations are also in progress to study the properties of the mutant and normal enzymes for comparison with those already delineated for the Negro and Caucasian subjects (Kirkman, 1959; Marks and Gross, 1959*b*).

Summary.

1. Deficiency of erythrocyte glucose-6-phosphate dehydrogenase has been demonstrated in the Papua-New Guinea population.

2. The trait has a much higher incidence in coastal areas than in the highlands — distribution which correlates well with malarial endemicity.

3. Possible quantitative differences in enzyme activity are discussed.

Acknowledgements.

Gratitude is expressed to Dr. D. C. Gajdusek, of the National Institutes of Health, Bethesda, Maryland, United States of America, to Mr. R. T. Simmons, of the Commonwealth Serum Laboratories, Melbourne, and to Dr. F. D. Schofield and Dr. B. Ryan of the New Guinea Medical Services for their kind cooperation in obtaining blood samples.

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PRE-NATAL SEX DETERMINATION.

By R. D. MACBETH, M.B., M.R.C.O.G.,
The Women's Hospital, Crown Street, Sydney.

IN the course of an investigation into certain characteristics of liquor amnii, it occurred to us that it should be possible to determine the sex of the fœtus many weeks before birth, from a study of the cells in the fluid.

We were obtaining liquor by abdominal amniocentesis, from the thirtieth week onwards, for another purpose; and we found that the fœtus lies in a fluid containing myriads of epithelial cells. Presumably these are being shed in a continuous shower.

By far the majority of these cells are degenerate, or have shrunken, pyknotic nuclei; but in each specimen there have been found a certain number of well-preserved cells with good nuclear detail.

Dr. J. Murray Moyes of The Women's Hospital, Crown Street, and Dr. Hirst of Sydney Hospital agree that the cells are mostly desquamated from the skin of the fœtus. There are others (James, 1956) who hold the view that the cells are largely from the respiratory and alimentary tracts and from the vagina of the fœtus. No appreciable difference in the numbers of squamous epithelial cells was noted between preparations from male infants and female infants, so we must conclude that epithelial cells from the vagina cannot be a significant proportion of the total.

These cells are the only portions of the baby available for study before birth, and it was on the advice of Dr.

[1] Read at a meeting of the Clinical Society of the Women's Hospital, Crown Street, Sydney, on April 5, 1961.

Murray Moyes that the investigation was turned to the chromatin content of these somatic nuclei. He has been of the greatest assistance in making the beautifully stained tissue sections and cytology smears on which this paper is based.

In 1924 Painter showed that in man the female carried a pair of like XX chromosomes, and the male an X and a smaller Y chromosome.

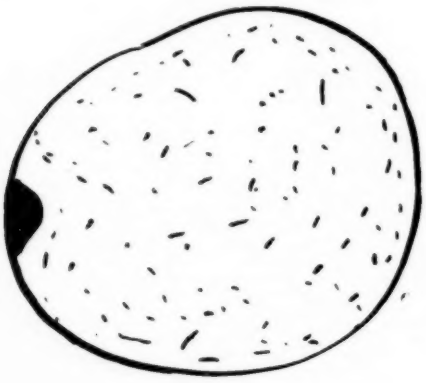

FIGURE I.
Sex chromatin mass lying against the inner surface of the nuclear membrane.

In 1949 Barr and Bertram discovered, while studying neuron cells, that in females a characteristic hyperchromatic mass was present in many nuclei. This was found subsequently in cells from most tissue, including skin, but indisputable hyperchromatic masses were not found in cells from males.

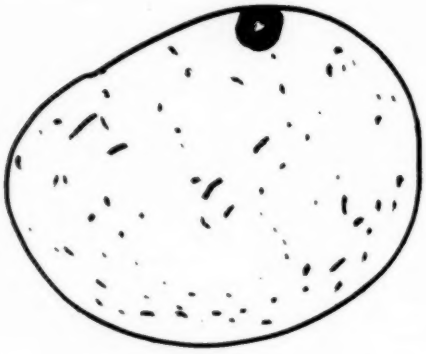

FIGURE II.
Sex chromatin mass lying against the inner surface of the nuclear membrane.

In the intermitotic or resting phase, the chromosomes lie scattered throughout the nucleus, apparently in a fragmented state. The larger portions can be seen when stained, but in the female there is in some cells a heterochromic mass, characteristically lying in contact with the inner surface of the nuclear membrane. This is thought to consist of regions of the two XX chromosomes remaining fused and compact in the resting intermitotic nucleus.

In the male cells the XY chromosomes do not produce a mass of comparable size or position in the nucleus.

This heterochromic mass has been called the "Barr Body" or "Sex Chromatin Mass".

The Barr Body needs some experience and much patience to find. In different tissues the percentage of readable nuclei varies from 10% to 50%, and only a small percentage of these would contain sex chromatin. It is well to make the criteria of acceptance very strict, or one will be accepting cells as sex chromatin-positive in error.

To be "readable" in the resting state the nucleus must satisfy several criteria. It must: (i) be clearly stained; (ii) be fairly large, with chromatin finely scattered (except the sex chromatin mass); (iii) not have a crenated membrane; (iv) not be folded; (v) not have stained particles overlying it.

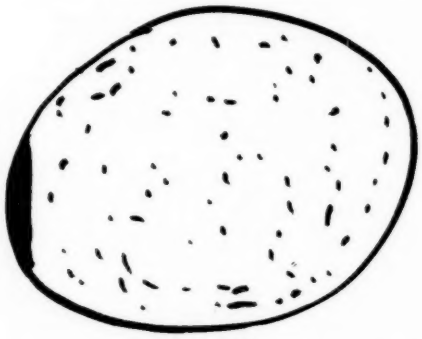

FIGURE III.
Sex chromatin mass lying against the inner surface of the nuclear membrane.

The sex chromatin mass is usually a single, clearly-outlined, planoconvex body on the inner surface of the nuclear membrane. This means one must view the nucleus

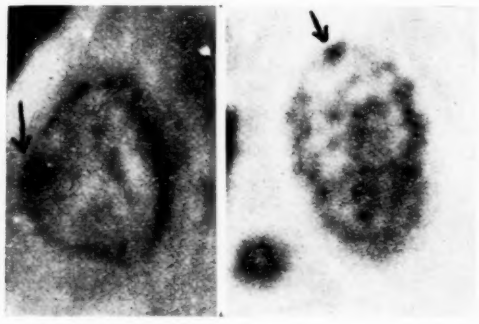

FIGURE IV.
Sex chromatin masses seen in nuclei of umbilical cords of female babies.

tangentially at the point where the mass lies. It happens therefore that many positive nuclei will be excluded, but errors due to foreign matter lying over the nucleus and to other nuclear inclusions are eliminated.

By focusing the microscope up and down through the nucleus under oil emersion one can determine the shape of the heterochromic masses. They are of a very deeply-stained, compact material — usually in the shape of a small oval nut — about 1μ in diameter. Acceptable variations are shown in Figures I, II and III.

The sex chromatin is Feulgen positive — a fact which is evidence of a high concentration of desoxyribose-nucleic acid.

It should not be confused with nucleoli or with chromocentres. These are larger and round, have a less sharp outline and are not usually against the nuclear membrane. In many of our films, cells with multiple nucleoli have

been seen; these cells have to be rejected or stained with the Feulgen reagent to select the sex chromatin.

In males there are often small, multiple masses of chromatin, but with experience one comes to recognize the true sex chromatin in the female.

The Present Work.

In order to be certain that sex chromatin was indeed being seen in cells from the liquor amnii, a comparable study was carried out of preparations made from buccal smears of adult and infant females. These smears showed about 20% of readable nuclei to contain sex chromatin masses.

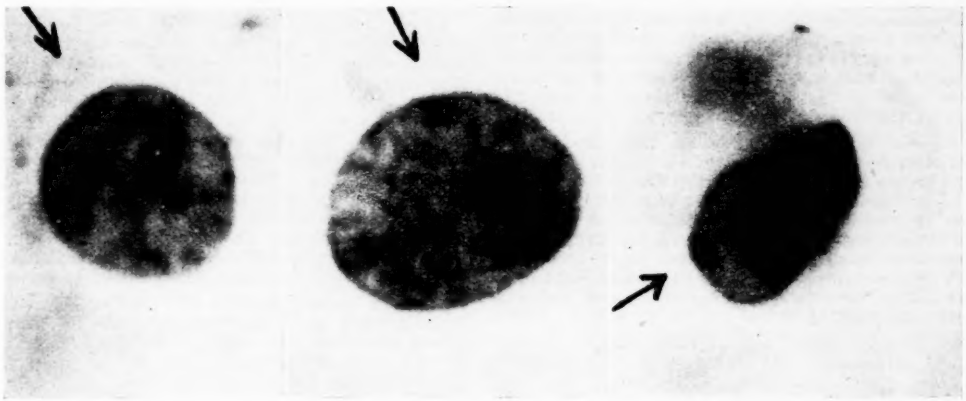

FIGURE VI.

Sex chromatin bodies seen in epithelial cells in liquor amnii obtained in cases of female babies.

Then preparations were made from the umbilical cords of male and female babies. This material is of course entirely of fœtal origin, and carries the chromosome pattern of the child. The best cells were found in the covering membrane of the cord, although some good nuclei were seen in connective tissue cells in Wharton's jelly.

For the study of liquor amnii the fluid was obtained by abdominal amniocentesis under sterile conditions; it was centrifuged, then a film was made, fixed and stained and the search was begun (Figure V[1]).

The difference between these films and the buccal films is that in liquor very few cells are well enough preserved to be readable. They lie in the midst of myriads of degenerate squames. Hundreds of cells are often scanned to find a few nuclei worth study, and only a few of these contain acceptable sex chromatin bodies. But the heterochromic mass has exactly the same characteristics in liquor cells as in other tissues, and when it is seen it is diagnostic of true nuclear female sex.

Because of the obvious difficulties in this method, another more accurate one was sought. Advances have recently been made in the studies and recognition of individual chromosomes. Ford (1958) described the basic steps in short-term culture of body cells *in vitro*. Mitosis is stopped in metaphase by the addition of colchicine; the preparation is treated with hypotonic solutions to swell the nucleus and spread out the chromosomes; and finally, by Ford's "squash technique", the chromosomes are separated before fixation, and in suitable preparations the individual chromosomes can be recognized. The X chromosome is found to be about seventh in magnitude, and the Y to be very small.

This matter was discussed with Dr. Peter Ilbery of the School of Public Health at the University of Sydney, who has worked with Ford. He has been attempting to culture cells from amniotic fluid, hoping to "keep them turning over", and then to stop them in metaphase and examine them for the presence of XX chromosomes. Unfortunately it has so far proved impossible to revive the desquamated cells in the amniotic fluid sufficiently to "keep them turning over". This work is continuing.

In some liquors of known female infants, there have been so few sex chromatin bodies found, that unless they are searched for with great patience they can easily be missed — and one should hesitate ever to be certain that a fœtus is sex chromatin-negative. If heterochromic sex chromatin masses are clearly found, then one can state that the child is female. But the implications, legal and social, which might follow a wrong prediction, make one determined to keep this as a research procedure for special circumstances.

[1]For Figure V see art-paper supplement.

Acknowledgements.

I wish to thank Dr. J. Murray Moyes of the Women's Hospital, Crown Street, for advice on the histology, and Dr. E. Hirst and the Photographic Unit at Sydney Hospital for the preparation of the very fine microphotographs.

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THE INCIDENCE OF SOFT-TISSUE INJURY IN AUSTRALIAN RULES FOOTBALL.

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Melbourne.

THE incidence of soft-tissue injury in Australian rules football is extremely high, although fortunately the serious injuries are rare. In any of the Melbourne league football clubs, of the 18 players who run out onto the ground for the first game, only a couple are likely to survive right through the season without losing a match because of injury. At Collingwood in the last six years, six players, although fit for normal, everyday work, since their injury have never returned to league football. Admittedly, two of these players were at the end of their football careers. During the season, five or six will require in-patient or out-patient hospital treatment because of football injury.

Broadly, these injuries fall into the following two main groups: (i) Those due to direct violence. These are more common later in the season, when the slightest weakness on the part of any player affects the team's prospects of winning the premiership. They may occur at the extremes of weather conditions, when the ground is stony-hard or when the ground is extremely soft. (ii) Those due to indirect violence. These occur most frequently when the player is unfit. They are, therefore, likely to occur early in the training period. It is interesting to note that in the 1960 Victorian Football League interstate team all the injuries were of the direct-violence type, none of the indirect type. All the players, of course, were fit athletes.

Of particular interest to anyone looking after footballers is the management of muscle injuries, as these form the commonest source of incapacity from football. It was in the management of these injuries that one found the most confusion amongst people treating them, and obtained the least help from the medical literature—and this in spite of the injuries occurring in epidemic proportions at this time of year.

Muscle injuries take the form of (i) torn muscle, due to indirect violence, and (ii) contused muscle ("corked leg", as the boys call it), due to direct violence. A study of a series of cases of each type of muscle injury was undertaken at Collingwood to establish some uniform method of treatment. To enable the series to be large enough, we have used the 250 or so hopeful recruits that each season begin training. In this series, 80 consecutive cases of torn muscle and 40 of contused muscle have been studied.

With any football injury, the burning question is not "what is the diagnosis?", not "what is the treatment?", but "how soon can I play football again?". This last question is asked by the coach and by the committee, and even the doctor's judgement is coloured by his desire to see a star player return to the side.

Torn Muscle.

Torn muscle occurs frequently in the pre-season training period, and implies that the player's preparation has been inadequate. The recruit is often very unfit compared with the trained athlete. At the beginning of the season there is, therefore, no difficulty in building up a very large series indeed. The condition is more commonly found in the player relying on his speed. It is seen, therefore, in the type of boy who can run 100 yards in 10 seconds.

A typical history is as follows. Usually the injury occurs early in the game, or early after half-time, when the player is not adequately warmed up. The boy is suddenly called upon to put maximal effort into the muscle. He feels a sudden snapping sensation in the region of the muscle, usually the thigh, sometimes the calf. This is followed by quite severe pain in the region of the muscle. The pain is made worse by any attempt to continue the exercise. When a calf muscle tears, one may have the sensation of having been struck over the back of the leg in the region of the tear. After rupture of the muscle, the boy is unable to continue the game. As he cools down, the pain becomes more severe. If untreated, the condition is followed by swelling in the region of the torn muscle. This swelling is made infinitely worse by any attempt to continue activity in an unlimited fashion. In a period of about five days to a week a bruise often appears in the region of the insertion of the involved muscle.

On examination of the patient immediately after the injury the following abnormalities are noted. (i) Pain is felt on attempting to contract the involved muscle. (ii) An area of extremely localized tenderness is found on palpation of the involved muscle. (iii) One may be able to feel a physical gap in the muscle. (iv) Later there is swelling and perhaps bruising. The local pain and tenderness may be abolished immediately by injecting a local anæsthetic agent into the tender area. Presumably the pathological basis is physical rupture of some of the fibres in a muscle bundle. This rupture would appear to be associated with a rupture also of a small blood vessel, as there is invariably some degree of swelling, and quite often an appreciable hæmatoma formation, as evidenced by the often pronounced bruising.

To establish a uniform method of treatment, injured players were divided into two main groups, each of 40. Those in Group I had absolute rest to the injured part until healing was sound; those in Group II had uninhibited activity, and were even urged to try early training. In each of these two main groups subgroups were given the following treatment: (i) local anæsthetic injections; (ii) injections of local anæsthetic agents plus hyalase; (iii) immediate pressure bandaging.

In all the cases the only factor we found influencing early return to full value football was rest. Injections helped neither the active nor the inactive group. Surely it is illogical to inject a substance into torn tissue, hoping it will speed repair. If the skin of the hand was incised, we would aim to hold the skin edges together and to rest the part until the skin had united. No one would consider injecting materials hoping to speed up the formation of scar tissue. In a healthy person, there is nothing we can do that will accelerate the rate of healing. All we can do is delay healing by inadequate or wrong treatment. In the group having absolute rest, the average time for their return to football was two to three weeks. In the active group, the average time was five to six weeks.

When a person returns to exercise before healing is complete, and the muscle breaks down, he commences his repair period all over again, from the time of this breakdown. Some of the boys in the active group managed to return within two or three weeks by being fortunate enough to avoid full stress on the muscle — that is, they did not break down and have to commence their repair period all over again.

Age influences the rate and sureness of healing. In the young player (under 20 years) healing is more rapid and sure than in the elderly player (25 years and onwards age group).

The following is an outline of treatment now used at Collingwood in the management of torn muscle. (i) We aim to prevent swelling from occurring, by such means as firm bandaging and the use of cold compresses and icebags, etc. (I doubt the value of icebags, but their use would appear to be traditional amongst footballers. They certainly are harmless.) (ii) We insist on complete rest to the part until healing is sound. Evidence of healing is (*a*) absence of pain and (*b*) absence of tenderness. (iii) We keep the remainder of the athlete fit, so that he may resume normal activities as soon as the injury has recovered. While he is recovering, the injured boy is handed over to the physical culture instructor. Any part not immobilized is very actively exercised. The injured player in fact trains harder than the boy out on the track. We must endeavour to keep his heart and lungs at their original state of efficiency, so that when the injury has healed he is able to resume full activity. Any sign of pain in the torn muscle is a danger signal, and exercises must be planned to avoid this pain, and therefore to avoid the risk of recurrence of injury. (iv) Probably the most important of all, we protect him from the greatest temptation with any athlete — the temptation to return to football too early. Nothing is more certain than that the player will break down if we allow him to put full stress on an unhealed torn muscle.

Incidentally, I do not believe that there is any difference in the recovery rate between torn muscle in a footballer and, say, that in a cricketer. A test cricketer, selected before complete recovery, is just as likely to break down in the first innings as a footballer in the first quarter.

The question of local anæsthesia has to be considered. Though not effective in aiding healing, local anæsthesia has one great value — that is, at the time of extreme

emergency, when there are no reserves to replace an injured player, and when winning or losing an important game depends on this player. Then, and then only, should local anæsthesia be considered and the boy be allowed to continue. Everyone knows that by continuing he is likely to lengthen his period of disability.

Contused Muscle—"Corked Leg".

The cause of contused muscle is severe direct violence applied to the muscle, usually the thigh muscle. As a result of the direct violence there is hæmorrhage into the muscle. I have seen one player's thigh increase in circumference by two inches in a matter of a quarter of an hour. The hæmorrhage on this occasion was severe enough to drop his hæmoglobin value to 80%. After the impact, the thigh feels numb and painful. For some minutes it is in fact useless. Those who have had a "corked leg" will understand how real a thing this numbness is — extending to the point of paralysis of the muscle involved. After some minutes the numb element wears off and the player is able to continue in a hampered sort of way. If the condition is untreated during the next 24 hours, the swelling becomes intense. The skin becomes stretched, as if over a drum. Within the next three to five days there is likely to be swelling also below the knee. Commonly the knee joint develops an effusion.

To convince the players of the desirability of immediate treatment, players were put into two groups until a series of 20 in each group had been studied. In Group I the boy attempted to play; in Group II he came off the ground immediately and underwent treatment. The footballer who played on lost an average one or two games. The player undergoing immediate treatment was able to play the following Saturday, or lost at the most one game.

We therefore see the desirability of immediate treatment, not just that day, not after the game, but immediately. This urgency of treatment offers the boy a prospect of returning to football one week earlier. Once again, the elderly player (over the age of 25 years or so) is much slower to respond.

To ensure an early return to football, and provided reserves are available, the boy with anything but the most minor of "corked legs" should leave the ground immediately and commence treatment as follows. A firm pressure dressing is applied to stop hæmorrhage from continuing into the muscle. The player remains on the couch in the medical room for at least one hour. During this time an ice-pack may be placed on the thigh, although I myself doubt its value. About an hour later the boy is allowed to have a quick shower, and after the shower the wet bandages are changed. Once again a pressure dressing is applied to the leg and he is not allowed to remove this pressure dressing under a period of 24 hours. After 24 hours the pressure is removed and gentle active exercises are commenced. The boy may gently massage his own thigh, or have gentle massage by a qualified physiotherapist ("gentle" being the operative word). Under no circumstances should the exercises or massage cause pain, as pain is an indication that further damage is occurring. The boy is warned of the danger of starting further hæmorrhage by too vigorous massage, or by too active exertion. Towards the end of the week there is often a residual firm area of hæmatoma in the thigh. Under these circumstances an injection of local anæsthetic and hyalase solution may be helpful. I am, however, unconvinced of its value, but at least it is harmless. The exercises are graduated until in about a week or ten days the boy is able to return to competitive football. Unlike the boy with a torn muscle, at this stage there is little likelihood of his breaking down — that is, if he is not again struck on the leg.

Once again, during the period of his incapacity he is handed over to the physical therapy instructor, who works out exercises involving everything but the injured thigh.

THE ROLE OF GASTRIC LAVAGE IN THE TREATMENT OF PATIENTS SUFFERING FROM BARBITURATE OVERDOSE.

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> The stomach should be lavaged well with warm water by use of the gastric tube to remove any remaining poison. (Goodman and Gilman, 1958.)
>
> In mild and moderate cases, if ingestion of barbiturates has been recent, gastric lavage should be performed. (Harris Isbell, in Cecil and Loeb, 1959.)

THESE are quotations from two well-known textbooks and are representative of the advice usually given in discussions on the treatment of patients suffering from an overdose of barbiturate drugs. However, the medical literature contains very few reports of analysis of gastric washings from such patients to justify this advice, and it is becoming increasingly recognized that gastric lavage in an unconscious patient may be a hazardous procedure.

Harstad, Moller and Simesen (1942) published the results of analysis of gastric washings in 80 cases of barbiturate overdose. In half of these cases, no barbiturate was recovered by lavage with 10 litres of water. In 86% of cases, less than 1·5 grains was recovered. In only two cases was more than 7·5 grains recovered. They noted that an average of 2·4 litres of fluid was retained by the patient, and they were able to demonstrate radiologically that some lavage fluid reached the lower part of the small bowel, and the caecum, before the lavage was completed. They suggested that some barbiturate might in fact be washed from the stomach into the small bowel by the first lavage fluid inserted. They could find no relationship between the amount of sedative recovered and the amount ingested, nor was the amount recovered related to the time since ingestion. They were unable to find reference to any previous attempts to evaluate the procedure, but noted that stomach lavage had been thought to be an essential part of the treatment of sedative overdosage since the beginning of this century.

I have been unable to find reference to quantitative investigation of the efficacy of stomach lavage since 1942, apart from Locket and Angus (1952) who washed out the stomach in all cases and found "large amounts" of barbiturate in some, even after four hours.

Eadie (1959) reviewed the cases of hypnotic overdosage at the Brisbane Hospital from July, 1953, to June, 1957. At that time there was no definite policy in relation to the use of gastric lavage. The development of a respiratory unit in the hospital provided an opportunity to assess certain aspects of the treatment of these patients, and since the middle of 1959 all unconscious patients suffering from hypnotic overdosage have been admitted to this unit. Two hundred and seventy patients have been treated to February, 1961, and two of these patients have died.

The Present Investigation.

Two major investigations have been carried out. One was to assess the rôle of "Bemegride" in reducing the mortality rate, as part of a controlled trial. The death rate has been so low that this investigation has been abandoned and replaced by a controlled trial to assess the effect of "Bemegride" in reducing the period of unconsciousness. These results will be reported later. The second investigation was to assess the rôle of gastric lavage, and the results of this investigation are now reported.

Material.

One hundred consecutive patients suffering from known or suspected barbiturate overdosage, who were unconscious on admission to hospital, were allotted to two groups as follows: Group I, patients known to have taken the overdose within three hours of admission; gastric lavage was performed on all of these; Group II, patients known to have taken the overdose more than three hours before

admission to hospital, or whose time of ingestion of the drug was not known. Gastric lavage was performed on alternate patients in this group. In addition, gastric lavage was performed on a further 15 patients who were conscious on admission to hospital. These constitute Group III.

Lavage was performed by a senior resident medical officer of the department of medicine or the department of anæsthesia. A cuffed endotracheal tube was inserted in almost all unconscious patients prior to passage of the stomach tube to prevent aspiration of gastric contents into the lungs. Six pints of warm water were used, the gastric contents being siphoned off after the insertion of each pint. All fluid returned was collected for analysis. Lavage was performed with the patient semi-prone and the foot of the bed elevated. The washings were analysed by the Queensland Government Analyst.

Results of Gastric Analysis.

Gastric lavage was performed on 76 patients, but the results of analysis are available for only 68. The washings from the other eight patients were discarded in error.

TABLE I.

Group.	Number of Patients.	Average Amount of Barbiturate Recovered (Grains).	Number of Patients with no Barbiturate in Washings.	Largest Amount Recovered (Grains).
I	25	3·4	4	17[1]
II	28	1·7	9	9·5
III	15	0·6	8	5
Total ..	68	1·8	21	17

[1] Lavage on this patient was performed by his local doctor soon after ingestion of the drug and prior to the development of unconsciousness. He was unconscious on admission to hospital and remained so for ten hours.

Complications of Gastric Lavage.

There were no complications in the group of conscious patients, and none due to trauma caused by passing either the endotracheal tube or the stomach tube. Complications did occur in the unconscious patients. Deeply unconscious patients with absent pharyngeal and laryngeal reflexes posed no problems in tracheal intubation and gastric lavage.

Patients with active reflexes reacted in a manner similar to that of some patients under light thiopentone anæsthesia. Temporary cyanosis developed in 10 patients as a result of the procedure. Laryngeal spasm occurred in nine of these 10 when tracheal intubation was attempted. Stomach washout was abandoned in three. After a period of rest, attempts were made to pass the stomach tube in the remaining six. Each had further laryngeal spasm as a result of pharyngeal stimulation, and five patients regurgitated stomach contents with some aspiration into the lungs. No attempt was made to intubate the trachea in the tenth patient. When the stomach tube was introduced into the pharynx, regurgitation of stomach contents occurred and severe cyanosis developed.

Results of Treatment.

One death in the 270 patients treated to the end of February, 1961, occurred in this series of 100 patients; the other was not in the lavage series.

The first was that of a man admitted to hospital 20 hours after the ingestion of pentobarbitone sodium. He was hypotensive and cyanotic on his admission. Stomach wash-out was not performed. He died 30 hours after admission from pulmonary œdema and purulent pericarditis.

The other death was that of a woman who was semi-comatose from an overdose of barbitone for five days before her admission to the respiratory unit. She was discharged to a psychiatry ward five days after that, able to speak; but she remained lethargic and depressed for another 10 days, when she died suddenly. Autopsy showed a considerable amount of barbiturate in her liver. She had chronic pyelonephritis and renal insufficiency.

Experience gained since the admission of these two patients suggests that earlier artificial respiration might have altered the outcome in the first case.

Gastric lavage has not been performed on any of the 120 patients treated since this investigation was concluded, and there have been no deaths in this group.

Discussion and Conclusions.

These results are very similar to those of Harstad, Moller and Simensen (1942). They indicate that in most cases of barbiturate overdose, stomach lavage removes only small quantities of the ingested barbiturate. There are definite hazards associated with stomach lavage in unconscious or semi-conscious patients, even in the conditions obtainable in an experienced respiratory unit. These hazards would probably be greater if stomach lavage was performed in smaller centres, or as emergency treatment in the home as suggested by Thomson and Alstead (1960).

In view of these findings, it is suggested that routine lavage of the stomach of patients unconscious as a result of barbiturate overdose should be regarded as potentially dangerous in all cases and of no value in most. I disagree with some present medical and lay opinion which suggests that failure to perform gastric lavage in all cases of poisoning by ingested barbiturate may constitute negligent treatment.

Summary.

Gastric washings from 68 patients suffering from barbiturate overdose were analysed. An average of 1·8 grains was recovered.

Of 61 unconscious patients, 10 became cyanosed as a result of the procedure. All of these 10 patients had incompletely depressed gag and laryngeal reflexes.

There has been no death in a further series of 120 unconscious patients suffering from sedative overdose since gastric lavage has been abandoned as part of the treatment.

Acknowledgements.

I wish to thank Dr. A. D. D. Pye, Medical Superintendent of the Brisbane Hospital, for permission to publish these cases.

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PERIPHERAL RELAXATION.

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Sydney.

FOR many years the relaxing agents have been used to prevent reflex movements during operations performed under light anæsthesia on sensitive areas of the body, especially the extremities. Despite the advent of trichloroethylene ("Trilene") and halothane ("Fluothane") they still have, largely for reasons of economy, a wide utility in this regard.

Yet, in such circumstances, the relaxing agents not infrequently fail to produce the desired conditions, and one must resort after all to deep general anæsthesia. The course of events is as follows. Anæsthesia is induced with thiopentone in moderate dosage; a tourniquet is applied

to the affected limb, and when the operation is about to begin the relaxant (decamethonium) is given in suitable dosage. General paralysis rapidly ensues, necessitating controlled respiration with nitrous oxide and oxygen, which might be expected to have some analgesic effect. Nevertheless, brisk reflex and other evasive movements now occur on surgical stimulation of the exsanguinated area, to the great embarrassment of anæsthetist and surgeon alike, while further doses of decamethonium are quite ineffective locally. A peremptory demand that the patient be "got under" now results in deep narcosis and delayed recovery, the latter being highly repugnant to current nursing opinion.

However, if the relaxing agent is given a few minutes before the tourniquet is applied, excellent operative conditions result. Further, such conditions will persist for as long as the tourniquet remains in place, a truly remarkable effect. But if it loosens, or otherwise becomes ineffective, reflex responses to surgical stimulation soon become troublesome, although now the administration of more relaxant should restore quietude.

The explanation of this disappointing response to decamethonium administration for long remained obscure, despite the obvious reason. No doubt the course of events would be similar if suxamethonium, gallamine or tubocurarine were being used instead. Ultimately it was realized that, since these agents act at the neuro-muscular synapses, any hindrance to their access would vitiate their efficacy there. Thus the application of a tourniquet to a limb before their administration would prevent such access, although the rest of the musculature is completely paralysed. Hence the relaxing agent is now given immediately after the induction of anæsthesia, following which a pause of from three to five minutes is observed before the tourniquet is applied. This is best done by the surgeon, largely for medico-legal reasons.

The prolongation of the relaxation is a striking and most useful feature of this technique. The part will remain immobile long after breathing has become restored; indeed it remains so for as long as the tourniquet is left *in situ*. Evidently no destruction of decamethonium occurs at the end-plates involved, while the arrest of local circulation effectively prevents its elimination from these sites. The only likely source of movement now is in the muscles proximal to the tourniquet, and these can be readily controlled either by deeper anæsthesia or by more relaxant, if necessary.

It is thought that the foregoing observations are of original character, since no mention of them has so far been encountered in the relevant literature. However, if the quotation of appropriate references has been omitted, due apologies are offered.

Reports of Cases.

MACROGLOBULINÆMIA AND SEVERE INTESTINAL HÆMORRHAGE IN LYMPHOSARCOMA.

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THIS case report describes massive intestinal hæmorrhage from lymphosarcomatous deposits in the small bowel in a patient with long-standing macroglobulinæmia. Resection of the lesion was required to arrest the bleeding.

Clinical Record.

In 1954 a man, aged 48 years, was treated with blood transfusion for severe hypochromic anæmia; his hæmoglobin value was 3·5 grammes per 100 ml. The fæces contained occult blood, but no source of intestinal hæmorrhage was found. Bone marrow and barium-meal examinations revealed no abnormality.

In 1956 the patient presented with weight loss, a dry mouth and swollen parotid, submandibular and lacrymal glands. The hæmoglobin value was 8 grammes per 100 ml., the total leucocyte count was 7300 cells per cubic millimetre, of which 14% were plasma cells. The bone marrow contained 10% of plasma cells. The serum iron level was 28μg. per 100 ml. and the serum gamma globulin level was 3·2 grammes per 100 ml. Macroglobulins were demonstrated in the serum by ultracentrifugation, and in plasma cells in the bone marrow by fluorescein-labelled antibody (Curtain and O'Dea, 1959). Biopsy of a submandibular salivary gland revealed lymphocytic infiltration. The patient was treated with blood transfusions and iron. Subsequently, in the belief that the condition might be a lymphosarcoma, 6μc. of radioactive phosphorus (^{32}P) was given over a period of three weeks. The salivary glands decreased in size and the number of plasma cells in the blood and bone marrow diminished.

In June, 1958 he presented with a red, indurated facial swelling. The cervical and axillary lymph nodes, the liver and the spleen were enlarged. The erythrocyte sedimentation rate was 130 mm. per hour (Westergren). A blood examination showed a microcytic hypochromic anæmia and rouleaux formation. The total leucocyte count was 4000 cells per cubic millimetre, with 9% "atypical lymphocytes resembling plasma cells". Platelets were seen in moderate numbers. The fæces contained occult blood. The Sia test for macroglobulins gave a positive result. The facial swelling had been treated with prednisolone (15 mg. daily) for two weeks, and subsided after a further two weeks' treatment.

In August, 1959, the parotid swelling recurred and the course of ^{32}P was repeated. Seven weeks later the patient had a sudden melæna. The hæmoglobin level was 4 grammes per 100 ml. and the platelet count was 120,000 per cubic millimetre. Laparotomy for continued bleeding revealed tumour infiltrating several coils of small bowel and the adjacent mesentery, lymph glands and retroperitoneal tissue (Figure I). This was identified as the source of bleeding, and several feet of bowel were resected. Histologically the tumour was a typical lymphosarcoma (Figure II[1]). The patient recovered slowly, and has remained quite well. In January and November, 1960, he had further parotid swelling which on the first occasion resolved with radiotherapy.

Discussion.

The clinical features of macroglobulinæmia are due partly to lymphoid hyperplasia and partly to the presence of circulating macroglobulins. The characteristic bleeding tendency is attributed to interference with various clotting factors, and damage to vascular endothelium by macroglobulin molecules (Kappeler *et alii*, 1958; Pachter *et alii*, 1959). Bleeding usually occurs from the oral or nasal mucosa. Massive intestinal hæmorrhage is rare in macroglobulinæmia and may be attributed to the bleeding tendency. However, the possibility of a local lesion causing hæmorrhage should not be overlooked. Cattan *et alii* (1959) record alimentary bleeding due to a benign gastric ulcer in a case of macroglobulinæmia. The bleeding was successfully treated by surgery. In the present case removal of a localized deposit of lymphosarcoma in the small bowel was life-saving.

In some cases of macroglobulinæmia no pathological basis is apparent, even after prolonged observation. Occasionally pathological macroglobulins may disappear spontaneously (Anderson and Ferriman, 1960). However, seven out of 10 cases of macroglobulinæmia reported by Mackay (1959) were associated with lymphosarcoma (this man is patient LAN in his series). Mackay believes that macroglobulins are synthesized by a mutant clone of lymphoid cells which may ultimately become neoplastic. The association of macroglobulæmia with malignancy has been demonstrated in mice with transplantable leukæmia (Clausen *et alii*, 1960).

[1] For Figure II see art-paper supplement.

This case illustrates the prolonged course of macroglobulinæmia and its association with lymphosarcoma, which was not fully confirmed until it caused severe intestinal bleeding. Removal of the bleeding lymphosarcomatous lesion was responsible for the patient's good recovery.

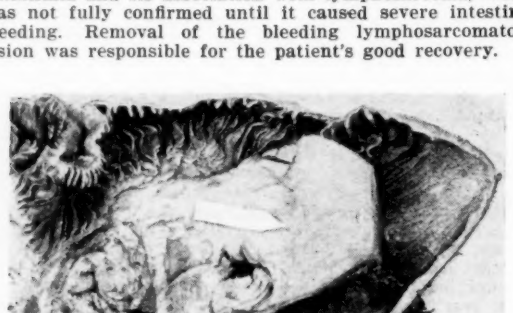

FIGURE I.

Operative specimen: lymphosarcomatous involvement of mesenteric lymph glands (white marker) and adjacent normal small bowel (half size).

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SIMULTANEOUS BILATERAL TUMOUR EMBOLISM OF THE COMMON ILIAC ARTERIES: A CASE REPORT.

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ALTHOUGH the concepts of thrombosis and embolism had been understood previously, it remained for Virchow to lay the foundations of our knowledge of the morbid anatomy of arterial embolism in a series of classical publications beginning in 1846 and in which the term "embolism" was first used. The first completely successful embolectomy (femoral) was achieved by Georges Labey of Paris in 1911.

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The sites of lodgement of emboli, and consequently the effects they produce, will depend primarily on their size. Jacobs (1959) has concluded that embolism is commonest in the smallest arteries and least common in the largest, but that this anatomical incidence is not evident in hospital series because many of the less serious cases do not come under observation. In the cases presenting, the common sites of lodgement are the aortic, femoral, popliteal and brachial bifurcations. An embolus temporarily lodged at one arterial bifurcation may become detached and lodge at the next lowest bifurcation, or an embolus saddled across a bifurcation may break into two halves, one of which may be carried into each trunk (Martin *et alii*, 1956). The latter part of this statement is believed to be demonstrated by this case.

Jacobs (1959) adequately demonstrated that the heart is the chief source of arterial emboli in his series of 122 embolic incidents. One hundred and twenty-one of these occurred in patients with heart disease, and in 98 (81%) there was an auricular arrhythmia of major type—usually auricular fibrillation, and occasionally flutter or paroxysmal tachycardia.

It is characteristic of malignant disease that tumour emboli are of microscopic size and consequently case reports of massive arterial neoplastic embolism are extremely rare. In 1940 Groth reported the first successful removal of a malignant embolus from the femoral artery of a female, aged 37 years, who later died of disseminated sarcomatosis. The tumour process had originated two years previously in the right tibia and amputation was performed. The embolus was then carried to the arterial circulation from pulmonary metastases. Till and Fairburn (1947) recorded a fatal case of femoral artery obstruction resulting in terminal gangrene, in which the embolus consisted of tumour cells from an occult pulmonary carcinoma. In 1950 Blum reported the second successful removal of a malignant embolus—from the left femoral artery of a male, aged 60 years. The patient died as a result of a primary pulmonary carcinoma after surviving the operative procedure by three months.

This case was thus considered worthy of report as the third recorded instance of successful removal of a malignant arterial embolus. Its added interest lies in the fact that two large emboli, presumably momentarily a single saddle embolus which broke, lodged simultaneously, one in each common iliac artery. Both were removed at the one operation.

Clinical Record.

Mrs. B., aged 52 years, presented with a five hours' history of pain of sudden onset, numbness and paralysis in both legs. There was a past history of two months' duration of progressively increasing shortness of breath with cough and wheeze, and one week before admission there had been a small hæmoptysis. Out-patient clinical and radiological chest investigations after the hæmoptysis had failed to establish any definite diagnosis, but a bronchial neoplasm was suspected.

On examination, the patient was seen to be a heavily built woman with a mild wheeze and dyspnœa, but no shock. Her pulse was regular and of normal rate; her blood pressure was 145/90 mm. of mercury and there was a moderate degree of cardiac enlargement, but no other evidence of cardiac disease. Clinically the respiratory system and abdomen were without abnormality. Both lower limbs from below the knees were white, cold and anæsthetic, and there was no peripheral circulatory return. The patient was unable to move her ankles or toes and all the pulses, including both femorals, were absent. The tourniquet test confirmed the absence of arterial flow in both lower limbs.

A diagnosis of saddle embolus was then made, but no theory was entertained as to its origin. The patient was submitted to immediate operation under general anæsthesia. A large right paramedian incision was made and the peritoneal cavity opened. Pulsation was noted at the aortic bifurcation extending distally to just above the

division of the common iliac arteries, where it ceased at approximately the same level on both sides. The peritoneum was incised in the line of the right common and external iliac arteries, and on gentle palpation an embolus could be felt at the right common iliac bifurcation. Three soft rubber tourniquets were applied in standard fashion well above and below the bifurcation and the embolus was exposed and found to extend distally into both the right external and the right internal iliac arteries. After careful extraction of the embolus and testing for its complete removal by the momentary relaxation of each tourniquet in succession, the artery was closed with a continuous atraumatic 0000 silk suture. Two thousand units of heparin were then injected into the common iliac artery above the suture line and at this stage in the procedure it was noted that all pulses had returned in the right lower limb. The pelvic peritoneum was now bluntly dissected up from the original incision in the posterior wall to reveal the left common iliac artery and its bifurcation, where a second embolus was palpated. This was then similarly exposed and removed and was found to extend into the left internal and external iliac arteries. Again 2000 units of heparin were injected and it was noted that all the pulses had returned in the left lower limb. The peritoneum of the posterior wall was closed without drainage and the wound was closed in layers.

Post-operative treatment included intravenous and oral anticoagulant therapy on routine lines, penicillin and streptomycin administration, breathing exercises and postural drainage.

Eight hours after operation the patient's general condition was good; sensation and movement were now present in both limbs, which were warm and pink and showed good circulatory return. However, the right leg was noticed to be cooler and weaker than the left. All pulses were normal in the left lower limb, but the right popliteal and dorsalis pedis pulses were now absent. On the first post-operative day the patient suffered a transitory episode of acute dyspnœa with epigastric pain, and the heart was found to be fibrillating. Aminophylline and digoxin were given and some hours later the heart rhythm became regular again and the patient felt well. She continued to make satisfactory progress, with pulses unchanged and both lower limbs clinically showing good circulatory return, until the sixth post-operative day, when the lower end of the abdominal wound burst. This was resutured immediately under a general anæsthetic, but the patient's post-operative condition was poor, as she had a combination of central and peripheral circulatory failure which failed to respond to treatment, and she died 24 hours later.

Autopsy Report.

Post-mortem examination revealed an extensive carcinoma arising from the main left upper lobe bronchus and replacing the whole of that lobe with necrotic growth. Both sides of the heart were dilated and the myocardium was flabby and pale. The sites of embolectomy were clear and free from hæmorrhage, but there was a recently propagated thrombus occluding the right internal iliac artery.

On macroscopic examination both emboli were seen to be Y-shaped and their largest (proximal) limbs had ragged ends. They had the appearance and consistency of organized thrombi (Figure I[1]).

Microscopic examinations of the lung growth and emboli were carried out. The lung growth was a typical oat-cell carcinoma with extensive areas of necrosis (Figure II). Sections taken through both emboli showed organized thrombus containing carcinoma cells indistinguishable from those of the pulmonary tumour (Figure III).

Commentary.

The likelihood of massive embolism resulting from a malignant growth is influenced by three main factors. The first of these is the rate of tumour growth. Rapidly proliferating anaplastic tumours have an increased likelihood of penetration of vein walls with minimal local thrombus production. The second factor is the size of the vein invaded by the neoplasm. Channels with a narrow lumen, when penetrated by growth, will suffer speedy occlusion with thrombosis on either side of the growth. However, with further progression of the growth and thrombus into larger channels the chances of detachment of a large tumour embolus become correspondingly greater. The third factor is the inherent tendency to intravascular invasion possessed by the tumour.

A combination of these factors seems to be responsible for certain varieties of neoplasm liberating tumour emboli with notorious regularity. These are chorionic carcinomata, anaplastic sarcomata, mixed tumours, hypernephromata and carcinomata of the lungs. Pulmonary neoplasm has been quite understandably responsible for all four reported cases of embolism of the systemic arterial system of the lower limbs. In this case the site of origin was patently the primary pulmonary carcinoma which was found *post mortem*, the cells of which were identical with those in the embolus.

Bronchogenic carcinoma commonly invades the pulmonary capillaries and veins (Macdonald and Heather, 1939). However, the tumour emboli so formed are usually of microscopic dimensions, owing to the constant respiratory movements and pressure variations causing detachment of tumour fragments from small veins and venules before any appreciable size has been attained (Willis, 1952).

In the light of both surgical and anæsthetic advances, and in accordance with the general principles of surgery, there is no doubt that early operation is imperative in arterial emboli threatening gangrene. Jacobs (1959) concisely states that "in limbs with adequate collateral circulation, signs of grave ischæmia do not persist for more than two hours after onset", and that "though the number of successes is not great, embolectomy holds first place in the treatment of embolism of the larger limb arteries".

Agreement has not been reached as to whether operation is indicated when gangrene is already present. Those in favour of operation feel that they will halt further progress of gangrene, thus allowing a lower amputation site, and that they will also obtain a more efficient function in the residual limb. The opposing view is that operation is unlikely to be successful owing to thrombus formation distal to the embolus. This will occur if the embolus has been present for a sufficient time to allow gangrene. Also it is technically more difficult to remove successfully an embolus that has been present for more than a few hours. It seems that these cases must be considered individually on their merits.

There remains the question, not truly applicable in this case, of whether such surgical interference is justifiable when tumour embolism has been diagnosed. The final answer to this question must lie with the surgeon and can be given only after studied appraisal of every patient as an individual. However, the following general facts should be taken into consideration, and should predispose the surgeon in favour of operative treatment. (i) Usually these patients are younger and physically more able to make the operation a success than are those whose emboli are cardiac in origin. (ii) The patients may be spared the pain and general distress associated with gangrene or the possible development of the tumour in the affected limb. (iii) The accepted principle of internal fixation of pathological fractures caused by tumour secondaries has recently set a precedent for a more radical approach to the problem of the patient with a hopeless prognosis.

Summary.

1. A case of bilateral iliac artery tumour embolus is described; its treatment and the post-operative course are detailed.

[1] For Figures I, II and III see art-paper supplement.

2. The literature regarding other cases of arterial tumour embolus is reviewed, together with theories of its ætiology.

3. Indications for operative treatment are discussed and a plea is made for operative treatment in those cases diagnosed sufficiently early.

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PILONIDAL SINUS OF THE OCCIPITAL REGION.

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THE term pilonidal sinus was first applied to a hair-containing sinus in the sacro-coccygeal region by Hodges in 1880, although the condition had previously been observed by Herbert Mayo in 1833 and described by Anderson in 1847 and by Warren in 1854. Hodges also mentioned the sole of the foot as a site of intrusion of short hairs from a mattress or mat.

The occurrence of hair-containing sinuses at other sites, and in particular the trouble caused by short hairs penetrating the skin between the webs of the fingers of barbers, were also noted by Hodges. Patey and Scarff (1946, 1948) and Ewing (1947) have described the lesions found in the webs of fingers as pilonidal sinuses, and Currie *et alii* (1953) showed that these are relatively common amongst hairdressers.

The occurrence of lesions at other sites has been described—in the anterior perineum (Smith, 1948), at the umbilicus (Jackson, 1854; Aird, 1952; Patey and Williams, 1956; Sadeghi-Nejad and Rains, 1958), the mons veneris (Thwaite, 1957) and the abdominal wall (Janelli, 1958).

MacLeod (1953) described a "pilonidal sinus" originating in a dermoid cyst in the suprapubic region. He suggested that such degeneration in dermoid cysts might occur elsewhere and these could be called pilonidal sinuses with some justification.

Currie *et alii* (1933) mentioned a case of an infected dermoid cyst of the nose rupturing, and resulting in a hair-containing sinus. However, they deprecated the use of the term pilonidal sinus for such cases, and for cases of sinuses between barbers' fingers in which ætiological and pathological factors are known. They suggested that the term pilonidal sinus should be used as it was originally used by Hodges to describe the post-anal lesion about whose ætiological and pathological factors there is still some doubt.

Clinical Record.

A boy, aged two years, was brought to hospital by his mother, who stated that she had first noticed a lump on the back of the child's head when he was only a few weeks old. Since then there had been a daily discharge of yellow material which the mother described as "pus". The child was inclined to scratch the area, and a scab had formed on the top of the lump. The child's health was otherwise good.

The child appeared healthy. There was a small lump 1 cm. in diameter on the occipital region of the scalp in the mid-line. A crust was present on top of the lump. An X-ray examination of the occipital region showed no bony abnormality.

The lump was excised through an elliptical incision, and the skin wound was closed. A histological report on the tissue removed read as follows:

> An ellipse of skin measuring 3·5 × 1·5 cm. was submitted. A small rounded elevation 7 mm. in diameter was present on the surface. In this there was an opening through which a tuft of hairs protruded. A second small opening was present elsewhere on the skin surface and again a tuft of hairs protruded.
>
> Histologically there was a sinus track lined by pyogenic granulation tissue, and associated with a fragment of hair shaft at the base. There were a few aggregates of chronic inflammatory cells in which pigment-containing macrophages were recognized. The features were those of pilonidal sinus.

The child has been followed up for three years. He has remained well, and there has been no recurrence of the lesion.

Summary.

A review has been presented of the sites in which pilonidal sinus has been recorded in the literature.

A case of pilonidal sinus occurring in the occipital region of a child has been described.

Acknowledgements.

We wish to thank Mr. Norman J. Logie for allowing us to publish details of the patient under his care, and Dr. Gordon Mathieson and Dr. R. C. Nairn for the histological report.

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Reviews.

The Rorschach Experiment: Ventures in Blind Diagnosis. By Samuel J. Beck, Ph.D.; 1960. New York and London: Grune & Stratton. 9" × 6", pp. 264. Price: $6.50.

HERMANN RORSCHACH'S "*Psychodiagnostik*" has attracted controversy since he published his "experiment" in 1921. Those who assert that human behaviour can be described

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only in mathematical terms reject it as being without significance, whereas others who know no bounds other than the limits of their enthusiasm are apt to substitute it for careful clinical appraisal. Evaluation of the test's accuracy in giving insight into the dynamics of a patient's personality is made the more difficult by the lack of measures against which the test itself may be tested; in other words, until personality theory is more developed than it is now, it is difficult to avoid arguing in circles.

Samuel Beck has tackled the problem from both directions in a series of contributions made during the last three decades. This book is his fourth monograph on the Rorschach test itself. It is not primarily concerned with his particular views on trait theories, but sets out to "demonstrate the processes entering into the interpretation of a Rorschach test protocol, the path the examiner travels from the raw data of the test . . to the clinical report which he turns over to the therapi[illegible]"

The intention is to show how well the test behaves as an objective instrument, and though the author's skill and integrity are demonstrated on every page, one also sees how difficult it is to interpret the test without mentioning extraneous data. Thus the interpretation of a woman suicide's record is an analysis in retrospect of why the patient suicided rather than a simple interpretation of the data. The author makes this distinction clearly enough, but this is not the purpose of the book.

The first part of the book is a discussion of Rorschach's *Erlebnistypus*, not only as a theoretical problem, but also in its relationship to such matters as the post-Bleulerian concept of pseudoneurotic schizophrenia. It is erudite and informative, although on the evidence available so far in this book and elsewhere, one may quibble about Dr. Beck's confidence that a satisfactory statistical evaluation of the problem will be reached.

Next, there is a section in which eight patients' records are analysed in detail; in a brief review one cannot but admire the skill with which it is done. We still cannot follow Dr. Beck in his scoring of M, even when he gives his reasons in detail, and we also think that the failure to score FM deprives a clinician of useful information.

Finally, the Rorschach correlates of neurotic defence mechanisms are discussed along with such important matters as treatability and the transference. No one who has wrestled with the problems presented by the Rorschach test can ignore this book.

Whillis's Elementary Anatomy and Physiology. By Roger Warwick, B.Sc., Ph.D., M.D.; fifth edition; 1961. London: J. & A. Churchill Ltd. 9¼" × 6½", pp. 282, with figures. Price: 24s. (English).

THE appearance of a new edition of this well-known textbook will be widely welcomed. For many years it has proved its value as an introduction to anatomy for medical students and as a textbook for nurses, physiotherapists and other medical auxiliaries. The last two editions have appeared under the able editorship of Professor Roger Warwick, who has preserved intact the original outline of the book, whilst making corrections of detail in the light of recent developments in anatomical knowledge. In this, the fifth edition, some new changes of this character have made their appearance, and there have been further small additions. A number of paragraphs treating of the more detailed matters have been reset in small print as a guide to the reader seeking only the most elementary account. The fifth edition of this book will continue to meet the many nee's for which it was well devised, and can be warmly recommended.

The Use of Isotopes in Hæmatology. By L. G. Lajtha, M.D., D.Phil.; 1961. Oxford: Blackwell Scientific Publications. 9" × 5½", pp. 92, with illustrations. Price: 21s. (English).

TRACER ISOTOPE STUDIES have added precision to hæmatological diagnosis over the past few years, and few hæmatology laboratories now do not possess some isotope equipment. Pernicious anæmia can now be diagnosed with certainty by the use of cobalt-labelled vitamin B_{12}, blood volume determinations are useful in the diagnosis of polycythæmia vera, and both ^{59}Fe and ^{51}Cr are employed in studying the kinetics of erythropoiesis and red-cell destruction. These tests have become routine to many hæmatologists.

This monograph sets out to offer a concise guide to the use of isotopes in hæmatology and is written primarily for the hæmatologist. The reaction of most hæmatologists should be one of disappointment, because the major part of the work is taken up with a description of methods with which he is familiar already. The hæmatologist about to embark on isotope techniques will find insufficient detail in the technical methods, and would be advised either to read larger and more detailed works or to consult the original papers in which these methods are published.

There are several misspellings, one formula is incorrect (page 24) and sentence construction is often loose and ambiguous. What does one make of this sentence which appears on page 48? "The proportion of the intravenous dose which appears is taken as the proportion of the absorbed dose which appears, hence the amount of absorption from the initial oral dose can be calculated."

The clinical interpretation of the results of isotope techniques could have been dealt with more fully. In particular, the section dealing with directional collimated scintillation counting over the surface projections of organs after the injection of ^{51}Cr-labelled red cells is very brief. This is a useful technique for the detection of splenic hypersequestration, and a decision concerning splenectomy may be made on the results of such tests. One paragraph only was allotted to this problem.

The two final chapters are worth while. One gives methods for labelling white cells and platelets with di-isopropylfluorophosphate, while the other deals with autoradiography applied to short-term marrow culture experiments. This latter chapter includes some beautiful plates from the author's original observations. There is also a table indicating radiation doses received in radioactive tracer tests.

It would be difficult to recommend this monograph as a useful addition to our knowledge concerning isotopes.

Cancer in Childhood and Youth. By Sigismund Peller, M.D., F.R.S.H.; 1960. Bristol: John Wright & Sons Ltd. 8¾" × 5½", pp. 304, with 29 illustrations. Price: 42s. (English).

THE content of this work is mainly epidemiological, and seeks to relate the salient differences between the incidence of adult and childhood cancers to theories of carcinogenesis of childhood tumours. This being so, we find the title far too sweeping, as it may needlessly attract the interest of clinicians, who will find little of value in this book.

The author tries to fit the epidemiological facts into one common mould—a unitarian concept of pre-natal exogenous carcinogenic stimulation as the basis of all childhood neoplasms. The main thesis runs along these lines: exogenous carcinogens are the predominant cause of cancer in man; this finds expression in the preponderance of epithelial tumours over tumours of vascular and connective tissues in adult life, the former being relatively exposed and the latter relatively sheltered from the exogenous carcinogenic stimuli; in childhood the reverse is true, the exogenous carcinogens reaching the fœtus via the maternal blood-stream, so that vascular and connective-tissue structures are relatively exposed and epithelial tissues relatively sheltered from inductive stimuli; this differential exposure to carcinogens results in the relative preponderance among childhood neoplasms of tumours of vascular and connective-tissue origin over those of epithelial origin, with malignant growths of reticulo-endothelial tissues being included in the former group.

This concept is elaborated in terms of the variations in vascularity and richness of blood supply to various organs, regions and tissues during fœtal development. The cranial end of the fœtus has circulatory advantages over the caudal end in the earliest weeks of development, but at the price of greater carcinogenic stimulation; the natural consequences of this differential stimulation are a relatively high incidence in childhood of tumours of the brain, and a preponderance of connective-tissue tumours of the cephalic end of the body over similar tumours in the more caudal regions. The author is indefatigable in adducing an enormous array of seemingly relevant data from cancer statistics, from embryology and from fœtal physiology to support the ramifications and elaborations of this concept in explanation of the specific tumour incidence in the various organs and tissues. When the known facts are grossly discordant with this unitarian concept, they are readily rationalized in terms of different degrees of latency of tumour induction in the specific tissues at fault, or by means of even more original concepts such as the release of locally carcinogenic substances during the normal physiological involution of the fœtal adrenal cortex.

In spite of the many qualifications and rationalizations which such a unitarian concept must inevitably require, the development of the main theme is often persuasive. The

history of experimental medicine has taught us that the value of imaginative inductive reasoning such as is indulged in in this book cannot be denigrated solely on the basis of lack of objective proof. The measure of whatever value this book may possess will be the provocation and stimulation it may afford to workers in the cancer field to explore the validity of the main hypotheses presented.

The concluding chapters of the book stray from the author's main theme into some of his thoughts on cancer immunization and a principle of inverse association which he developed in a previous work ("Cancer in Man", 1952), but these detract from rather than add to the interest of the book as a whole, which will probably be limited to those working in the field of cancer research.

Psychoanalytic Concepts of Depression. By M. Mendelson, M.D.; 1960. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications Ltd.; Toronto: The Ryerson Press. 9" × 6", pp. 180. Price: 52s. (English).

THE concept of depression, from being a relatively simple one, has with increasing study become so complex that psychiatrists and psychoanalysts alike are in considerable confusion about it. In this work the author aims at studying "in a meaningful critical way" the development of the Freudian concepts of depression. He also gives some attention to diagnostic problems and the difficulties of definition.

The introductory chapter giving the general history of the use of the word in psychiatry is a very good one. The opinions of Kraepelin, Meyer, Kirby, Bleuler, Aubrey Lewis and lesser authorities are summarized, and the perennial issues, organic versus psychological cause, psychotic versus neurotic and endogenous versus reactive, are well brought out. One is left with a fairly clear picture of the present-day position.

When it comes to giving an account of the contributions of the psychoanalysts, the author is less at home. A large number of papers, both psychoanalytical and otherwise, are referred to, and the subject is dealt with both chronologically and in respect to specific issues; but one does not obtain a clear perspective. The author states that his text was expanded from a brief paper, and this no doubt explains why it is disjointed and hard to follow. Almost the whole of page 119 is an exact repetition of what occurs on pages 51 and 52, and one has the general impression of scissors and paste rather than of a masterly gathering together of the many divergent views.

The author's recognition of the semantic confusion is good, but far from complete, and it is clear that until there is some standardization of terminology there is little point in trying to reconcile all the contributions made by psychoanalysts and others to this difficult subject. There are only a few misprints in the book.

Pneumoconiosis: Modern Trends. Report of Meetings held in Birmingham (April, 1959) and in Glasgow (January, 1960); 1961. London: The Chest and Heart Association. 8½" × 5½", pp. 144, with illustrations. Price: 18s. 6d.

EIGHTEEN contributions, two of which present information not as yet available elsewhere, averaging about 2500 words each, make up this slim volume. The chapters are of somewhat uneven standard, as one might expect, and the two chapters on respiratory physiology involve unnecessary repetition inseparable from the mode of presentation. An excellent summary of "modern trends" in the field of pathology is given by E. J. King, though we do not subscribe to his dogmatic over-simplification of the clinical diagnosis of silicosis, "recognized in life by the shortness of breath of the patient, particularly on exertion, and . . . diagnosed by the X-ray film", which is misleading on at least two counts. P. Hugh-Jones politely produces his customary competent survey of lung function, expertly modified to suit each new audience, in the course of which he utters a fundamental truth which clinicians cannot heed too carefully—that the relation of lung function to radiographic change involves the study of a random population. Without a background of this experience, physicians and radiologists making any deductions as to lung function—and hence inevitably as to prognosis—from the radiograph of a penumoconiotic patient are treading on very dangerous ground, for the population they normally study is heavily biased. Several of the authors, particularly Stewart Rae and A. Meiklejohn, stress the vital importance of good films and meticulous technique, especially when periodic reexamination is contemplated. Incidentally, it is remarkable, in view of the admitted difficulties and limitations of periodic X-ray examinations and the uncertainty as to what advice should be given the individual in consequence, that no mention is made of periodic estimation of ventilatory capacity. This may well prove more important and informative in the long run, and the results, especially in conjunction with the radiographs, must influence the physician's approach to the individual worker. Other notable chapters deal with rehabilitation, notably in a "closed workshop" (A. A. White), morbid anatomy (A. G. Heppleston), tuberculosis and pneumoconiosis (R. I. McCallum) and the less common and more recently recognized pneumoconioses (A. T. Doig). Whilst the discussion of treatment is open to some criticism, there are some interesting observations on what the worker should be told as to his radiographic status and his prognosis and as to the desirability or otherwise of changing his job. The approach to these problems is conservative and lacks only an assessment of function as well as radiographic shadows to be reliable. Mr. William Ure, of the Amalgamated Union of Foundry Workers, in usefully discussing the industrial welfare of pneumoconiotic patients, refers to a Russian foundry where anyone exposed to dust was free to go at any time to a special room for steam inhalation treatment. "I know nothing of its prophylactic or therapeutic value *but* [the italics are ours—it is such a notoriously familiar "but" to industrial doctors] I . . . found it was not unpleasant and seemed to have a freshening effect on the respiratory organs"; which goes to show that the Russians are as far ahead in industrial psychology as they are in making rockets.

Much of this book deals with British conditions; but it may be warmly commended to doctors in industrial or general practice as an easy and informative evening's reading. The expert should at least glance through it for the sake of the widely different perspectives it contains, although he will learn more from other recent publications in this field. The book is well produced, though conservative book-lovers will not appreciate the style of the title page, and no one can forgive the omission of the titles of papers from the references, a lazy habit which appears to have been copied from some weekly medical journals where at least there is the excuse of space. The X-ray films and figures are excellent, although it is not clear why Professor Meiklejohn's vital slides were omitted and Professor King was allowed to slip into a book of this size and character a reproduction of two pages from the first English edition of Ramazzini (1705).

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Neural Mechanisms of the Auditory and Vestibular Systems", edited by G. L. Rasmussen, Ph.D., and W. F. Windle, Ph.D., Sc.D.; 1961. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9¾" × 6¾", pp. 422, with illustrations. Price: £6 6s. (English).

"Recurrent Dislocation of the Shoulder", by H. F. Moseley, M.A., D.M., M.Ch. (Oxon.), F.R.C.S. (Eng. and Canada), F.A.C.S.; 1961. Edinburgh, London: E. & S. Livingstone Ltd. 11" × 8½", pp. 164, with illustrations and stereoscopic viewer and five Viewmaster stereoscopic reels. Price: £6 net (English).

"Field Studies in the Mental Disorders", edited by Joseph Zubin, Ph.D.; 1961. New York, London: Grune & Stratton. 8½" × 5¾", pp. 496. Price: $6.75.

"Strangulation Obstruction", by Isidore Cohn, jr., M.D., D.Sc. (Med.); 1961. Springfield, Illinois: Charles C. Thomas: Oxford: Blackwell Scientific Publications. 9¾" × 6¾", pp. 274, with illustrations. Price: 94s. (English).

"The Physics of Radiology", by H. E. Johns, M.A., Ph.D., F.R.S.C., LL.D.; second edition, 1961. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9¾" × 6¾", pp. 768, with illustrations. Price: £9 4s. (English).

"Teaching of Psychiatry and Mental Health", by M. Bleuler, O. V. Kerbikov, E. E. Krapf, T. A. Lambo, S. Lebovici, Tsung-Yi Lin, I. Matte-Blanco, M. Ostow, T. F. Rodger, J. Stoetzel and E. S. Turrell; World Health Organization Public Health Papers No. 9; 1961. Geneva: World Health Organization. 8½" × 5½", pp. 186. Price not stated.

"Control of Soil-Transmitted Helminths", by P. C. Beaver, World Health Organization Public Health Papers No. 10; 1961. Geneva: World Health Organization. 8½" × 5½", pp. 44. Price not stated.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 23, 1961.

VITAMINS GALORE.

In the history of scientific discoveries of great importance to medicine, the names of Eijkman, Funk, Hopkins, MacCallum, King, Darby and many others as pioneer workers with vitamins hold honoured places. Not only did the work of each bring to a fitting climax the labours of the many who had preceded them in their particular line of study, but each made a significant contribution towards the alleviation of a group of diseases which have, through man's history, claimed millions of lives. It has been estimated that during the three centuries from 1500 to 1800 more sailors died from scurvy than from all other diseases, wounds and disaster at sea, and that in 200 years from 1600 to 1800 over a million perished because of scurvy. It is, however, interesting to reflect that cures for the main vitamin deficiency diseases were known and applied long before the concept of a vitamin was developed, and thus long before a concentrated or synthetic form was available. It appears that in ships of the East India Company, oranges and lemons or lemon juice had been used regularly as antiscorbutics from 1601 onward, and it is well known that Lind had demonstrated upon a British man-of-war that scurvy could be cured in six days with two oranges and a lemon daily. Likewise in 1882, Takaki[1], the Director-General of the Medical Department of the Japanese Navy, had shown that beriberi could be both cured and prevented by increasing the daily allowance of vegetables, fish, meat and barley. Similarly physicians in England and the Scandinavian countries were using cod-liver oil for the treatment of rickets and osteomalacia in the first quarter of the last century.[2] In these times with an abundance of synthetic vitamins, it is noteworthy that Gomez and his co-workers in Mexico[3] have demonstrated that they can cure severe malnutrition in children with a generous diet without vitamin supplements.

The discovery of the chemical structure of the vitamins and their subsequent synthesis by the organic chemist have made available, in pure form, relatively enormous quantities of most of the vitamins. Having produced this plenitude, the pharmaceutical industry has the task of disposing of the surplus in an era of relative abundance of foods and a steady but definite improvement in the diet of the great mass of the population, at least in the economically advanced countries. The enrichment of cereals with vitamins offered one outlet, and it is now an accomplished fact that all flour milled in the U.S.A. is enriched. We understand that an appreciable amount of flour used in Australia for the manufacture of special breads, for which a premium price is charged, is now fortified with a number of vitamins of the B group. Attempts have, apparently, been made to extend this to a greater percentage of the Australian flour,[4] although the evidence for signs due to deficiencies of these vitamins in the Australian population is, at best, extremely meagre.

Of more immediate interest to the medical profession is the suddenly augmented vitamin-selling campaign by a number of pharmaceutical houses in Australia. This in itself could be disturbing; but of greater concern are the indications of a quantity or value race, which is suggested by the nature of the claims for some products, in that they appear to be based largely on the number of vitamins and minerals they contain, whether or not there are any indications that the healthy individual needs additional amounts of many of them, or on the magnitude of the dosages, whether or not these bear a resemblance to the daily requirements for the maintenance of health. It is hard to find evidence of the need for the Australian population to be taking most of these vitamins as pharmaceutical products. Unfortunately, also, much of the advertising material is misleading, since it appears to be based on statements about the functions of the particular vitamins long since discarded by the scientist.

Vitamin A is an example of the grounds for concern on both these counts. The requirements for this vitamin have never been as clearly defined as has been done for some of the water-soluble vitamins, perhaps because of the capacity of the liver for storage. The most extensive scientific work in this field is that done at Sheffield during the last war. From the results of these experiments, it was concluded that the minimum protective dose of vitamin A for adult males is 1300 I.U. per day, but a figure of 2500 I.U. daily was recommended to cover individual variations and to leave a margin for safety.[5] The current figure recommended by the National Research Council of America[6] is 5000 I.U., where the vitamin A activity is supplied by vitamin A and beta-carotene. It would appear that some pharmaceutical firms have misread these figures, for they are offering preparations of vitamin A which provide 5000 I.U. or more daily — and this for a population whose intake of vitamin A and carotene from dairy products and vegetables is in the vicinity of 7500 I.U. per day. The requirements for infants and children have been set by various authorities at levels between 1100 and 3000 I.U. per day. Some authorities have used body weight as the guide to determine the intake recommended; the manner in which other figures were determined is not clear. An infant or child who receives a balanced diet in which milk is the main source of protein will receive somewhere between 1100 and 2000 I.U. per day, and so far as we are

[1] Harris, L. J. (1938), "Vitamins and Vitamin Deficiencies", Churchill, London.

[2] Hess, A. F. (1929), "Rickets Including Osteomalacia and Tetany", Lea & Febiger, Philadelphia.

[3] *Amer. J. Dis. Child.*, 1954, 87: 684.

[4] National Health and Medical Research Council, 1959, Special Report Series No. 9.

[5] *Spec. Rep. Ser. Med. Res. Coun. (Lond.)*, 1949, No. 264.

[6] *Nat. Res. Coun. (U.S.A.)*, 1958, Publication No. 589.

aware signs of vitamin-A deficiency have never been recorded in an Australian infant. Additional vitamin A provided through a pharmaceutical product must then be surplus and would be available for storage in the liver. The capacity of the liver to store vitamin A without harmful effects has not been determined; but hypervitaminosis A is a well-described clinical entity, arising from intakes in one child of 460,000 units daily for six weeks, and in another of 280 units daily for two years.

Recently in this Journal there appeared an article by F. W. Clements, W. B. Macdonald and H. E. Williams[7] drawing attention to the possible dangers of large doses of vitamin D obtained by infants and children because of the unnecessary enrichment of cereals and other foods with vitamin D. It is gratifying to be able to report that the manufacturers have since withdrawn vitamin D from these products, leaving pharmaceutical products as the dominant oral source of vitamin D. However, pharmaceutical products designed specially for infants and children are appearing with doses well above those recommended by the National Health and Medical Research Council. For these reasons we believe the medical profession, and particularly pædiatricians, have cause for concern, since one of these products is such that to be effective for its prime object, it should be given to children continuously for upwards of ten years. Practically every child who would be given this product could be expected to obtain his daily requirements of vitamin A through ordinary foods. Thus it is possible to calculate that, on the assumption that all the additional vitamin A will be stored, something like 18,000,000 units will accumulate in the liver in this time. The effects of this must, at this stage of our knowledge, remain a matter for conjecture, but the thought is disturbing.

The appearance of vitamins B_6 and B_{12} in a number of products in the last few years is a further example of painting the lily. So far as we have been able to discover, there is no substantial evidence that the healthy infant or child eating an ordinary diet has any need for additional amounts of either of these vitamins. Surely, the main function of the token amounts of substances like choline and pantothenic acid in vitamin preparations for the aged must be to improve the appeal of the product rather than to improve the nutrition of the consumer. A review of the scientific evidence available indicates that the only vitamin supplements which have been shown to be useful in the routine management of infants and children are vitamin C and vitamin D, and possibly vitamin K in the new-born. The great majority of the products are meant to be over-the-counter sales for the apparently healthy child, and not to be solely dispensed on a medical prescription for the malnourished or convalescent child.

The physiological and pharmacological properties attributed by a few pharmaceutical firms to some vitamins are, to say the least, suspect; for example, it has been stated that vitamin A protects against colds and other infections. This idea is apparently based upon the earliest experimental work with this vitamin, when the observed changes in experimental animals were misinterpreted. A large number of subsequent experiments have shown that vitamin A given in excess of dietary requirements does not in any way protect the individual from infection. Similarly claims are frequently made that ascorbic acid will also protect against infection; the evidence to support this claim is extremely meagre, and such as there is seems to depend upon the observation that in individuals with infections the plasma ascorbic acid level is low. This seems to be an example of the result of a process, rather than the cause. It would appear that some machinery is needed in Australia to set a guide to the limits of the amounts of vitamins that should be included in preparations designed largely for over-the-counter sales. A need also exists, too, for a guide to acceptable claims for the beneficial effects that might be expected from self-administered vitamin preparations. These would be of use to the medical profession and of considerable benefit to the general public. With the strong element of competition that seems to exist in this branch of the pharmaceutical trade, it does not appear that the industry itself can be relied upon to do this.

It would be unfortunate if the conditions in Australia duplicated those in America, where, apparently, the public is reputed to spend some $350,000,000 a year on the purchase of vitamin preparations. This state of affairs prompted a physician[8] at the American Medical Association Congress, held at the end of 1960, to point out that "the vitamin manufacturers really ought to start processing sewage; they have been so successful in selling the American public superfluous vitamin pills, . . . that American sewage must now contain the world's richest concentration of vitamins".

[7] MED. J. AUST., 1961, 1: 236 (February 18).

[8] *Science*, 1960, 132: 1750 (December 9).

Comments and Abstracts.

TREATMENT OF CHORIONCARCINOMA BY IMMUNOTHERAPY.

IT has long been realized that chorioncarcinoma in the female may regress after the removal of the primary tumour, and it has been suggested that immunological factors might account for this phenomenon. In 1958 I. Doniach and his colleagues[1] reported an attempt to treat a patient with chorioncarcinoma by immunization with her husband's cells. They pointed out that the tissue of a mother and child are not compatible. Post-gestational chorioncarcinoma is unique in that it has its origin in placental tissue and therefore owes its existence in part to the germ plasm of the consort. It can therefore be considered analogous to a graft from the offspring to the mother. The result of Doniach's attempt was inconclusive, for though the patient experienced a gratifying remission of symptoms for a time, there was a subsequent local recurrence, and shadows suggestive of metastases appeared in the lung fields.

A group of Canadian workers (B. Cinader *et alii*[2]) now report a second case in which, after unsuccessful chemotherapy, subsequent treatment was confined entirely to immunotherapy. The patient, aged 21 years, had passed a hydatidiform mole, and one month later X-ray examination revealed metastatic deposits in the lungs. Hysterectomy was performed and the diagnosis of chorioncarcinoma was confirmed. Chemotherapy with vincaleukoblastine sulphate produced no definite change. The patient then

[1] *J. Obstet. Gynæc. Brit. Emp.*, 1958, 65: 553 (August).

[2] *Canad. med. Ass. J.*, 1961, 84: 306 (February 11).

underwent a course of active immunization against her husband's leucocytes. During the next three months the concentration of chorionic gonadotrophins in the urine fell steadily, and there was a diminution of the metastatic deposits in the chest. The patient was then given 10 ml. of gamma globulin prepared from a rabbit immunized against her husband's spermatic fluid. This produced a sharp reaction, but this was followed by a progressive improvement in her condition. There was a temporary rise in the level of chorionic gonadotrophin in the urine, but thereafter this fell steadily to normal, where it has since remained. Seven months after treatment the patient was well, and chest radiographs showed no evidence of metastatic disease. The authors conclude by stating that the possibility of spontaneous regression must be considered, but they point out that the first signs of improvement coincided with immunotherapy, and suggest that such treatment is worthy of further trial.

In discussing the theoretical basis for this form of treatment, the Canadian authors point out that chemotherapy has on several occasions been successful in the treatment of post-gestational chorioncarcinoma, but has been a failure in the case of male chorioncarcinoma. They suggest that this may be due to partial destruction of the tumour liberating substances which, in the case of post-gestational chorioncarcinoma, may initiate an antibody response which completes the eradication of the tumour. Because of the fœtal nature of the tumour, chorioncarcinoma presents a special problem, and immunotherapy in this case should not be confused with the more general problem of developing auto-immunity against cancer arising from the patient's own tissues, a line of research which is at present being actively pursued in a number of centres.

POSTERIOR PITUITARY ANTIDIURETIC HORMONE AND TOXÆMIA OF PREGNANCY.

THE ætiology of toxæmia of pregnancy continues to provide a prolific field for speculation and research, but in spite of the amount of attention that the question is receiving, it continues to prove an intractable one. In last year's William Blair-Bell Memorial Lecture of the Royal College of Obstetricians and Gynæcologists, M. Lindsay Paterson[1] of Glasgow took the opportunity to present an account of his studies on the rôle of the posterior pituitary antidiuretic hormone in toxæmia of pregnancy. The pressor or antidiuretic hormone and the oxytocic hormone are present in the posterior pituitary lobe and are liberated in response to stimuli such as variation of the osmotic pressure of the blood, suckling, emotional stress, shock, hæmorrhage and certain drugs. However secreted, the antidiuretic hormone acts on the kidney causing inhibition of water diuresis without change in the blood flow through the kidney. Paterson discussed the methods of extraction, identification and assay of the antidiuretic hormone from urine and blood plasma employed. By the use of two-dimensional paper chromatography, the urine of toxæmic pregnant patients was found to contain a substance which took up a characteristic position on the chromatogram. Commercial abstracts of posterior pituitary lobe and extracts of human posterior pituitary lobes showed a similar substance in the same characteristic position. It was then found that only that portion of pituitary extract which contained the substance under investigation demonstrated antidiuretic activity. Various animal and human experiments suggested that the antidiuretic hormone was present in the blood and urine of preeclamptic patients. In each case the substance was chemically a member of the 14-amino acid group, and was an active principle of the posterior pituitary lobe. Investigations of the blood and urine of normal women during the phases of the menstrual cycle showed no evidence of the presence of antidiuretic hormone. However, the hormone could be detected in the second half of the menstrual cycle in patients with premenstrual tension. The appearance of the hormone coincided with or preceded the weight gain noted by patients. Normal pregnancies and the early puerperium were not as a rule associated with antidiuretic hormone in the urine or plasma. In patients with preeclamptic toxæmia, antidiuretic hormone was present in the urine and blood when clinical œdema was moderate or marked. Six eclamptic patients admitted to hospital after having had at least one fit showed antidiuretic hormone persisting in the blood and urine for some time after delivery, the disappearance of the hormone coinciding with the disappearance of the œdema. Various experiments on alterations of osmotic pressure of the blood and altered concentrations of sodium, potassium and chloride did not elucidate the cause of altered antidiuretic hormone content of the blood. Mention is made of the possible rôle of the adrenals in the ætiology of preeclamptic toxæmia and also the suggested synergic action of antidiuretic hormone and oxytocic hormone of the posterior pituitary lobe.

The foregoing is a brief and somewhat superficial résumé of a very thoughtful contribution on the problem of toxæmia, one which is, moreover, based on a considerable body of careful experimentation. Paterson cauticusly permits himself to conclude that two facts have been demonstrated — namely, that there exists in the blood and urine of preeclamptic and eclamptic patients an antidiuretic substance which is not found in the blood or urine of normal pregnant patients, and that this antidiuretic substance is in fact the antidiuretic hormone of the posterior pituitary lobe.

[1] *J. Obstet. Gynæc. Brit. Emp.*, 1960, 67: 883 (December).

AIR POLLUTION.

AT first glance, the study of urban air pollution may appear to be a rather narrow specialty; but in reality it is an extremely wide subject, with aspects touching diverse fields. Engineers, chemists, physicians, meteorologists, plant physiologists, lawyers, veterinary scientists and economists are all concerned with the over-all problem. Because of this diversity, most of the virtual flood of literature on the subject now available deals with particular aspects, and general treatments are exceedingly few. For this reason, a new volume in the World Health Organization's monograph series should make a welcome addition to many libraries.[1] It contains 14 contributions from acknowledged experts in all the fields mentioned above. Each chapter in the book consists of a review of existing knowledge of the particular aspect covered, and in most cases the contributions are well documented. To ensure that the subject matter faithfully reflected world trends, the individual chapters were submitted to a panel of experts which met in Geneva in 1957. This procedure apparently delayed publication, as there are few references later than that year; but the book cannot be regarded as out of date.

The chapter on the effects of pollution on human health by H. Heimann is very comprehensive, and is followed by nearly ten pages of references. Very little has been omitted, and useful accounts are given of the various major pollution episodes involving loss of human life. The effects of the individual contaminants, likely to be present in urban air, are also described, though in some cases the author is irritatingly vague. Thus, under sulphur dioxide, he refers to the effects produced by "a rather low level of concentration" and "somewhat higher concentrations", and then "still higher concentrations". Though the reader is given references for further information, it should have been possible to include some idea of the actual concentration ranges in the text.

Some of the other subjects included are "A Historical Review" (E. C. Halliday), "The Rôle of Meteorology" (H. Wexler), "Physical and Chemical Aspects" (M. Katz) and "Control of Air Pollution by Site Selection and Zoning" (J. R. Taylor, A. Hasegowa and L. A. Chambers). It is

[1] "Air Pollution", by various authors, World Health Organization Monograph Series, No. 46: 1961. Geneva: World Health Organization. 9½" × 6¼", pp. 442 with illustrations. Price: £2.

interesting to note from the last-mentioned chapter that the situation confronting Los Angeles and vicinity, in which general circulation patterns produce prolonged subsidence inversions, also occurs frequently on the east coast of Australia.

This book is a useful source of authoritative information on the subject of air pollution, which is assuming increasing importance each year.

SHORTER ABSTRACTS.

GYNÆCOLOGY AND OBSTETRICS.

THE TECHNIQUE AND COMPLICATIONS OF AMNIOCENTESIS. A. W. Liley, *N.Z. med. J.*, 1960, 59: 581-586 (December).

THE author reports the results of 200 consecutive amniocenteses performed at the Postgraduate School of Obstetrics and Gynæcology, Auckland. Of the failures he attributes 10 to an anterior position of the fœtus and 6 to an anteriorly-placed placenta, and concludes that the periumbilical area is the most fruitful for puncture. Most patients were treated as out-patients and none were premedicated; reassurance was found quite effective in ensuring the patient's immobility, which in turn was found to be the best preventive against puncture of blood vessels. Although this complication gave little trouble in the present series, accounting for transient peritonism in four cases, whenever aspirate was contaminated with blood its source (fœtal or maternal) was determined by alkali-denaturation tests.

The most serious complication was infection, which occurred in two cases, both with fatal results for the fœtus, although the mother responded well to antibiotics. The incubation periods were 5 and 7 days respectively. When pregnancy was normal except for suspected hæmolytic disease of the child, which was ruled out by amniocentesis, no premature labours followed the procedure (44 cases). When abnormality existed, such as multiple pregnancy, hydramnios or severe fœtal hæmolytic disease, the high incidence of premature labour was thought to be due to the abnormality. When premature labour occurred in cases of antepartum hæmorrhage without placenta prævia it was thought that the hæmorrhage itself was probably a warning of subsequent events.

DETECTION OF RECURRENCES OF CANCER OF THE CERVIX UTERI BY REDUCING SUBSTANCES. A. Perroy and R.-A. Guérin, *Presse méd.*, 1961, 69: 1221 (June 3).

THE authors state that the discovery of cancer cells in vaginal scrapings is the earliest evidence of the existence of a uterine tumour or of the local recurrence of a treated lesion, provided that the tumour is "open" in the vagina. They consider that the level of altered reducing substances in the serum should be regarded as a routine test in the follow-up investigation of patients treated for cancer of the uterine cervix. It appears to be of considerable value when there is a recurrence of a tumour having no communication with the vaginal cavity (parametrial extension). However, significant changes in the serum level of altered reducing substances occur only when the tumour has grown large enough to produce general metabolic disturbances. In the authors' opinion, this is why these two totally different tests should both be used.

MEDICAL HYPNOSIS FOR OBSTETRICS. Frank Maya and L. Stanley James, *J. Amer. med. Ass.*, 1960, 174: 2026-2032 (December 17).

THE authors state that acceptance by the American Medical Association of medical hypnosis, and its reception by some as an ideal form of analgesia and anæsthesia in obstetrics, have given impetus to the collection of objective data. They present a survey aimed at comparing the acid-base status of infants born with the aid of hypnosis alone with that of infants born with the aid of various analgesics. The criteria of selection were designed to eliminate every variable factor except that of analgesia. In the three control groups the mothers had (i) caudal or pudendal block anæsthesia, (ii) caudal or saddle block anæsthesia with premedication, and (iii) cyclopropane anæsthesia with premedication.

The infants were evaluated clinically by the Agpar method and by the "time to sustained respiration". The serial biochemical tests were performed on cord blood and included estimations of oxygen saturation, carbon dioxide content, pH and buffer base content. The results indicated that clinically the babies from the hypnosis group were in better condition than those born with the aid of cyclopropane and premedication, but not significantly better than the other two groups. At birth all babies were slightly or moderately asphyxiated, and the biochemical tests revealed that the recovery of the hypnosis babies was more rapid than that of any of the other groups. It is mentioned that if the hypnotist is unskilled the mother may produce tetany in herself by hyperventilation and also lower her own blood oxygen tension by profound bearing down, and that both these conditions can affect the child.

THE ONSET OF STRESS INCONTINENCE. W. J. A. Francis, *J. Obstet. Gynæc. Brit. Emp.*, 1960, 67: 899-903 (December).

THE author presents the results of a study on the urine-passing habits of 400 unselected pregnant patients (222 primigravidæ and 178 multigravidæ) attending a hospital ante-natal clinic. The particular problem under investigation was stress incontinence, its incidence and its time of onset. Two hundred and sixty-eight of these women had stress incontinence and were subjected to cystometry. Eighty-three patients (33 with stress incontinence during pregnancy and 50 with good bladder control) were investigated by urethrocystography. These 83 women had further urethrocystography two days after delivery. One hundred and eighteen primigravidæ (53%) and 150 multigravidæ (85%) experienced stress incontinence during pregnancy. However, 42% of the primigravidæ had occasionally suffered loss of urinary control prior to pregnancy. The author observed that stress incontinence rarely, if ever, appeared for the first time after childbirth, but commenced either before or during pregnancy. Of the multigravidæ with stress incontinence, 40% noted this condition for the first time in the pregnancy under observation; 47% had had incontinence in a previous pregnancy with relief after delivery; 13% had had stress incontinence during and apart from previous pregnancies. None of the multiparæ had suffered stress incontinence for the first time during a previous puerperium. The condition had always commenced while they were nulliparous or during one of their pregnancies. It was noted that stress incontinence, once present, tended to recur in a more marked degree in each subsequent pregnancy. Ultimately the condition remained after delivery and became a persistent complaint. The time of onset of stress incontinence during pregnancy had an equal incidence in the first, second and third trimesters. Having occurred during pregnancy, stress incontinence usually increased in severity until term. None of the women investigated developed impaired bladder control for the first time after delivery. Pelvic floor exercises with the use of a perineometer on 24 patients with stress incontinence after delivery did not produce noticeable clinical or radiological improvement. X-ray studies by urethro-cystography showed absence of the posterior urethro-vesical angle in 90% of patients with stress incontinence seen during pregnancy. This finding corresponds to the accepted X-ray appearances in stress incontinence of the non-pregnant patient. The author noted that the posterior urethro-vesical angle did not appear immediately after delivery even though the patient recovered bladder control. The anatomy of the urethra and bladder returned to its preparturitional state almost immediately after labour. It is concluded that those women who develop stress incontinence in middle life are destined to do so from an early age, but that pregnancy, rather than parturition, reveals the defect and makes it worse.

LYMPHATICS OF THE VULVA. E. Parry-Jones, *J. Obstet. Gynæc. Brit. Emp.*, 1960, 67: 919-928 (December).

THE author reports reinvestigations of the lymphatic system of the vulva, questions the correctness of long-accepted anatomical teaching in this respect and discusses his findings in the light of radical surgery for carcinoma of the vulva. He observes that the vulva is continuous with adjacent skin, but developmentally has no connexion with it. A diffusible dye — Patent Blue V or Evans Blue — was injected into the subcutaneous tissues at varying sites and in different directions as described. Spread along lymphatic vessels was noted visually and at operation on the vulva which followed. The author describes the lymphatic vessels from the clitoris, vestibule, labium minus, labium majus, perineum and skin adjacent to the vulva. His results are illustrated and summarized as follows. Lymphatic vessels

from the vulva do not cross the labio-crural fold at any point to reach their termination in the inguinal and femoral nodes. The lymphatics of the labium majus travel along the length of the labium. Lymph flows readily from the labium minus to the labium majus, but not in the reverse direction. Lymph flows readily from the labium minus to the vestibule, but not from the vestibule to the labium minus. The lymphatics from the vestibule go alongside the clitoris or to the posterior part of the labium majus. The lymphatics from the perineum travel either immediately outside the vulva or along the labium majus. The lymphatics of the skin immediately adjacent to the vulva go into or run parallel with the labio-crural fold. No evidence was seen for or against contralateral or retrograde spread. In view of these findings, the author considers it not unreasonable to alter the surgical approach in radical vulvectomy to conform with this concept of lymphatic drainage. Since direct lateral spread of lymphatics to the thigh is not evident, Parry-Jones believes that an incision extending beyond the confines of the vulva should be avoided except in cases in which growth approaches the labio-crural fold or involves the perineum. This restriction of the vulval incision enables the wound to be closed without tension. The advantages which accrue from primary closure of the wound are emphasized. The lower portion of the urethra should be removed, not only when it is involved by growth, but also when the growth involves the labium minus.

THE SMALL FULL-TERM INFANT AND PLACENTAL INSUFFICIENCY. W. J. Rumbolz *et alii*, *West. J. Surg.*, 1961, 69: 53-60 (March-April).

THE authors present a review of 57 cases of placental insufficiency resulting in small, undernourished, full-term infants, which was communicated to the Eighth Congress of the Pan-Pacific Surgical Association in 1960. Placental insufficiency is described as a condition in which the placenta is unable to support the fœtus in such a way that growth and development take place at a normal rate. The cases under discussion comprise an obstetrical entity in the third trimester of pregnancy which continues to term, or near term, without normal increase in size of the uterus, in which puny babies weighing no more than 2050 grammes are delivered. The incidence of this condition in four Nebraska hospitals was 0·24% of 17,049 deliveries. Placental insufficiency is thought by the authors and others to be due to gradual obstruction of the maternal blood supply by the deposition of fibrin within the intervillous space, by thrombosis of maternal vessels or by degenerative lesions in the intima of the smaller uterine arteries. Clinically the condition should be suspected when failure of the uterus to enlarge is noted during the last trimester. Eighteen of the 57 patients had toxæmia of pregnancy (31%), including preeclamptic toxæmia, toxæmia superimposed on essential hypertension, and previously existing cardio-vascular-renal disease. Uterine growth was normal to about 31 weeks and then failed to continue at a normal rate. Mention is made of recent investigations into placental insufficiency by arteriography and estimates of urinary pregnanediol excretion. The over-all fœtal mortality rate was 40% (nine intrauterine deaths, six deaths during labour and eight deaths in the immediate post-partum period). Eight of the 23 fœtal deaths occurred in patients with toxæmia of pregnancy. The authors consider that a toxæmic patient presenting failure of uterine enlargement during the last trimester of pregnancy has a poor prognosis for fœtal survival. The question is raised as to whether such patients should have labour induced at the thirty-eighth week. The surviving infants in cases of placental insufficiency soon gain weight and follow normal growth patterns. There were six instances of congenital anomalies in the series (10·5%). Fifteen placentas were of normal size and 26 were small. They all looked abnormal, with areas of infarction and fibrosis in varying degrees. The authors recommend that delivery in a suspected case of placental insufficiency should be conducted in the same manner as a premature delivery, with no sedation prior to delivery, oxygen inhalation and conduction anæsthesia.

SEPTIC ABORTION WITH VASCULAR COLLAPSE. G. V. Anderson and M. Kadner, *West. J. Surg.*, 1961, 69: 129-131 (March-April).

THE authors discuss the lethal effects on and suggested management of patients with severe sepsis and vascular collapse with infected abortions. There were 21 deaths from this condition at the Los Angeles County Hospital during the two-year period 1956-1958 out of a total of 6787 abortions. Despite early diagnosis and modern therapy, including electrolyte and fluid balance studies and administration of antibiotics and vasopressor drugs, there is probably a mortality rate of 50% when septicæmia and vascular collapse are present. Significant post-mortem findings in these patients were pulmonary œdema and metritis. Retained placental tissue was found in 35% of fatal cases. A review of 14 additional deaths reemphasized that patients succumbed from vascular collapse after an initial favourable response to vasoconstrictors. Gram-negative bacilli cultured from the uterus and/or blood were most sensitive to chloramphenicol. Prevention of septicæmia and vascular collapse is stressed. The authors recommend adequate and proper antibiotics at the proper time, early evacuation of the septic uterus and the maintenance of adequate tissue perfusion. They postulate the following body changes in such infections. Bacteriæmia causes an initial vasoconstriction which, when prolonged, leads to inadequate tissue perfusion, with subsequent ischæmia, increased permeability, vasodilatation and irreversible shock. In some instances pressor substances may exaggerate the action of bacteria or endotoxin on the arterioles and venules. Patients dying of septic shock have normal adrenal output of steroids; but the level of cortisol concentration may be inadequate protection against tissue injury of this nature. The authors consider that steroids are valuable adjuncts to treatment. The following routine plan of treatment has effected a considerable reduction of deaths in these cases. On diagnosis, as a routine cultures are taken and the patient is given 1 gramme of chloramphenicol in 200 ml. of fluid by intravenous infusion. Subsequently 4 to 6 grammes are given per 24 hours. Blood and/or plasma is given to restore or maintain blood volume, and immediate evacuation of the uterus is performed. Two hundred milligrammes of hydrocortisone were administered intravenously at once, and 1200 to 1500 mg. of cortisone were given during the first 24 hours. The authors are unable to state whether massive antibiotic therapy, early uterine evacuation or steroids have been most beneficial.

BREAST CANCER IN PREGNANCY. T. L. Montgomery, *Amer. J. Obstet. Gynec.*, 1961, 81: 926-933 (May).

A SURVEY of the breast carcinoma problem in Philadelphia showed that, in a period of 10 years, there were 70 cases of cancer of the breast detected either during pregnancy or in the six months immediately after its termination. These cases constituted one in 70 of the total reported, and one in nine of the patients aged under 40 years was affected. Consistently throughout the series, the disease was found by the patient herself (90%), in spite of the fact that most of these patients were coming regularly to the obstetrician for pre-natal care. In 60% of cases there was failure to examine the breast, or misinterpretation or belittling of the physical finding, resulting in delay in tissue diagnosis. In the first five-year series, metastases were present, in 74% at the time of operation; 20% of these were systemic in distribution, and 45% of this group were dead in less than two years and 66% within five years. One significant breast lesion is likely to be encountered in every 100 pregnant patients previously unscreened for breast disease, and one in 10 or less of these lesions will prove to be cancer. Biopsies of nodules which appear during pregnancy are made under local anæsthesia, and if cancer is detected, the author recommends prompt termination of the pregnancy with or without ablation of the ovaries, preliminary X-ray therapy and radical mastectomy. Early and significant lesions of the breast can be detected in pregnancy only if the physician makes an irrevocable rule of including breast examination at each pre-natal and post-natal visit.

GERMAN MEASLES IN PREGNANCY. H. I. Kantor *et alii*, *Amer. J. Obstet. Gynec.*, 1961, 81: 902-905 (May).

THE authors report the outcome in 92 confirmed cases of rubella in pregnancy in Dallas, Texas. Of these cases, 72 occurred in the first trimester; 55 of these pregnancies resulted in full-term deliveries, with 47 normal babies, unaffected by the disease. Of the eight remaining babies, three had defective hearts, two had cataracts, two were stillborn (one as a result of placenta prævia), and one was found to be deaf at the age of nine months. Of the fœtuses exposed to the virus during the first trimester, 10% were aborted spontaneously early in pregnancy; of those which were carried to term, 11% had congenital abnormalities, and 4% were stillborn. When rubella appeared in the second or third trimester, all the babies were normal.

The Australian Scene.

SOME PROBLEMS ENCOUNTERED IN THE AERIAL TRANSPORTATION OF PATIENTS TO HOSPITAL.[1]

IT is a great honour for me and for the Royal Flying Doctor Service that I should be asked to deliver the John Alexander Cameron memorial lecture for this year. Dr. Cameron was not known to me personally. I knew of him as an outstanding doctor who had dedicated the greater part of his life to the pursuit of healing and counselling the sick in Ipswich. During the course of the preparation of this talk, I have made many inquiries of men who knew him and had the pleasure and privilege of working with him regarding his character, his professional skill and his many lay pursuits. To be able to enter into the proper spirit of any memorial lecture, I think that one must first have a comprehensive mental picture of the man himself who is so greatly honoured by his colleagues after his death; one must also appreciate the ideal behind the institution of any memorial that perpetuates an individual's memory in such a distinguished and practical way.

Dr. Cameron was a tall, courtly and softly-spoken man and a deft, meticulous surgeon. Above all, he was a loyal and helpful comrade to his fellow practitioners, and during his life he was responsible for cementing firm and lasting professional relationships with the medical men of Ipswich and Brisbane. This camaraderie has survived him, and that in itself is a monumental legacy. If I was asked now what I consider to be the most important attribute of a great doctor, I should say without hesitation that it is unselfishness. Medicine over the ages and in whatever plane demands absolute and unqualified unselfishness. The well-being of the patient takes precedence over every personal consideration, and the treatment of one's colleague in all the facets of professional relationships is no less a responsibility. Tonight my humble endeavour is to add another memorial to a man who was cast in the heroic mould of selflessness in the cause of humanity. No talk on the Royal Flying Doctor Service would be complete without some reference to its founder and early history. We have all heard and read of John Flynn, and most of us know that he was a missionary who kept the flame of Christianity kindled in the country where living was hard and churches were few. In John Flynn's travels he was witness to many a tragedy brought about only because no medical aid of any shape or form was available. He saw many a life needlessly sacrified—like Stoics, they lay and died. I have often wondered how this man of God felt in the face of such needless suffering and loss. I, too, have wondered whether it was any one incident that fired him with the tireless tenacity he displayed in bringing this great service into being. One thing we do know for certain, that it was as far back as 1912 that he first made known the substance of his dreams of combining medicine, aviation and radio as being the only hope of bringing solace and security to the inlander. All of us here tonight will have some appreciation of the state of aviation and radio communication in those years prior to the first World War, and we must concede that he was at grips with an almost impossible task. To John Flynn, however, nothing was impossible; from what I know of him he had one great attribute to which many of his "avowed victims" can testify—he never took "no" for an answer. The next 16 years of his path were strewn with innumerable and difficult obstacles, not the least of which was finance.

First, there was the Great War to end wars, which, though it hindered his plans at the time, at least was responsible for the first tremendous advances in aviation and radio communications. In the years following the war he worked tirelessly towards his aim, and with the help of Alfred Traeger, Dr. George Simpson, Qantas Airways and his few financial supporters, Flynn saw an aircraft depart on its first medical emergency mission from Cloncurry in May, 1928. Few people shared his confidence and delight on that day; but Providence ordained that this great man of God should live to see the tiny seed of his imagination and zeal blossom to the full flower of practical realism.

The development of the pedal radio transceiver is one of the great romances of the early history of this service; but times does not permit me to give any details of the many heartbreaks and triumphs that Alfred Traeger experienced in the nurturing of his radio child. Suffice it is to say that under the guidance of his genius and initiative, advances in radio communications have kept pace with the developments in medicine and aviation, and today the Traeger transceiver is in the forefront of world radio equipment.

Our first base established at Cloncurry in the far north-west was the only base in Queensland until the Charleville Base, at which I now work, was opened in 1945 by Dr. Allan Vickers. Nine years later our third base was opened at Charters Towers. This last-mentioned base serves mainly the Cape York Peninsula. The 600,000 square miles of Queensland are adequately covered at the present time by the three bases already mentioned.

Let me say a word about the remainder of the continent's inland. Scattered throughout the length and breadth of our 3,000,000 miles there are now 13 bases of the Royal Flying Doctor Service—six in Western Australia, three in Queensland, two in the Northern Territory and one each in New South Wales and South Australia. There are approximately 1600 outpost radio transceivers providing the communication link with those 13 bases. These radios also provide the means whereby the scattered settlers communicate with each other. This is a great personal comfort particularly to the women, who lead such lonely lives—they are of inestimable value as a medium of social intercourse over vast distances, they provide that feeling of nearness and security in the hearts and minds of these people who, by their courage, toil and spirit, are responsible for forming the core of Australia's national character. A school of the air is also carried on at some of our bases, and this service relieves the already over-burdened mothers of the tedium of teaching the children themselves. Each outpost is provided with a medical chest, which contains a number of drugs and instruments used for the routine treatment of the ailments commonly encountered. These medical chests are kept up to date by a medical subcommittee of the Federal Council, which meets each year to discuss advances and amendments.

Throughout the last year some 1500 patients, 500 of them aborigines, were transported to hospital for urgent medical treatment. About 500,000 miles were flown in these errands of mercy and routine care of the inlanders. More than 10,000 patients were seen by our doctors on their routine clinical flights.

A word about the very important matter of finance. We rely to a great degree on voluntary subscription, principally from those people who depend on the service. These subscriptions are subsidized by the State Governments in varying proportions — our Queensland Government has always been very generous. Added to this we receive a Federal capital grant, which is divided between the States at the discretion of the Federal Council of the Service.

Next let us glance at the following figures, which refer only to Queensland. I have covered the period between 1953 and 1960, mainly because it is over these years that I have been intimately connected with the service as one of its doctors. These figures portray a very definite pattern of change. The number of flights undertaken and the mileage flown have increased about 25%, from 98,000 miles and 230 flights in 1953 to 132,000 miles and 330 flights in 1959. As against these figures, the number of patients seen at our routine clinics and the number of immunizations carried out have increased approximately 500%, from 2000 clinic consultations in 1953 to 10,000 consultations in 1959, and from 94 immunizations in 1953 to 6000 in 1959. Granted that many of the immunizations were the result of

[1] The John Alexander Cameron Memorial Lecture, delivered on July 15, 1960, at Ipswich.

the widespread Salk vaccination campaign, nevertheless the greater number were the result of an increased awareness by the bush people of the importance of prophylactic medicine.

The Royal Flying Doctor Service is gradually changing from what was one time a purely emergency service to what is now principally a clinical service. When all of Queensland and the neighbouring Northern Territory was covered only by one aircraft operating from Cloncurry, there was naturally very little time available to the doctor to carry out routine clinical work. When the Charleville Base was established in 1943, much of the load of emergency work was taken over by the new base, and Dr. Vickers at Charleville instituted the first routine clinics at Thargomindah, Windorah and Jundah.

With the establishment of the Charters Towers Base in 1952, still more of the emergency work was apportioned out, and urgent flights were naturally shorter. This led to the establishment of clinical flights from both Cloncurry and Charters Towers, and to the extension of the clinical flights already being carried out at Charleville. At the present time in Charleville, between 300 and 400 routine consultations are being carried out each month. Approximately the same number of patients are being seen at the other two bases.

In the late twenties, and even up to the end of the last war, there were very few privately-owned aircraft in the outback. The picture has changed — there are many privately-owned aircraft nowadays, there are small charter companies operating from all the larger western towns, and the Channel, Gulf and Peninsular Services being operated by the two major airlines provide an avenue of quick, comfortable transport for the inhabitants. Added to this, roads are much better and luxurious sedan cars are plentiful. Also, in the last decade or so we have seen the establishment of aerial ambulance bases in Queensland, particularly along the coastal belt. The point I want to make is that few people should suffer acute hardship for the want of aerial transport to hospital at the present time, and upon this statement develops the nucleus of this lecture.

Quite apart from the increasing importance of routine medical care of the inland population which I have outlined earlier is the importance of a doctor and his aeroplane. Even patients with comparatively minor illness can be made much more comfortable with the judicious use of the many drugs available to us. The pilot's job is made immeasurably easier and safer when he can calculate the advisability of a flight in marginal conditions against the risk of a delay to the health of his patient. Only one person can assess the urgency of a patient's illness, and that person is the doctor and no other. A glance at our record over the past 32 years makes this fact abundantly plain. We have had one disaster, and even then the patient was uninjured.

As you probably all realize from reading our frequent advertisements for doctors in the Journal, we have some difficulty in recruiting and maintaining medical men in our service. I have often wondered why—our salaries and conditions of employment are perhaps better than those offered in similar full-time medical appointments. Some time ago I had occasion to interview a doctor who had just submitted his resignation, and I asked him why. I was particularly interested, because this man was also interviewed by me when he applied for the job. He said: "Firstly, because too much of my time is wasted in just flying, and secondly, I feel that my colleagues in the city are getting ahead of me in the education race to become specialists, and after all, nobody in his right senses wants to die a G.P.".

Nine times out of ten—nay, perhaps 99 times out of 100—the doctor considers that flying time is wasted, even though some of us consider that it could be put to profitable use in catching up with our journals. But that other time, how necessary it is for us to be there! I can recount many such experiences.

"Is it necessary for us to go, Doctor? The weather over the ranges is bad—thunderstorms—and the weather at base is doubtful." The patient was an old lady with acute intestinal obstruction and a host of anxious relatives. Gastric suction, a sedative and an "intravenous drip" kept us all happy overnight, and we made an uneventful return to base in the morning. Imagine the scene without the doctor—the patient groaning, the relatives pleading and the pilot worrying. Perhaps he could have made it—how many of them have not! Our service is more expensive, but we have not had to replace valuable aeroplanes and priceless personnel.

The answer to the second question—is the Flying Doctor wasting his time professionally? To quote directly from a statement made by Dr. Allan Vickers in an address delivered at the annual meeting of the Queensland Branch of the British Medical Association in 1958:

> He is in reality engaged in general practice, except that he uses radio and an aeroplane where the conventional practitioner uses the telephone and a motor-car.
>
> But we recognize that our doctors are isolated from the stimulus of regular contact with a teaching hospital, and therefore we allow, in addition to recreation leave, a period of four weeks on full pay each year, during which a refresher course is taken at an approved hospital. . . .
>
> Apart from this, the doctor will have the satisfaction of doing a job which is of unique value to his country: he will have as patients the finest and most grateful people in Australia; and he will be working for a friendly, cooperative organization which uses a minimum of red tape.

Now that I have stressed at some length the first point, that of operational safety from the flying point of view, in getting the patient to hospital, I should like to quote some cases from memory in which the safety of the patient from the medical point of view was at stake.

It was only some 18 months ago that I was called one late afternoon to attend a patient who had accidentally shot himself. The bullet entered the right side of the neck and ricocheted off the second cervical vertebra, then amputated his uvula neatly, deflected off his hard palate, fractured the ramus of his mandible and finished its trajectory under the skin in front of his left ear. When I saw him, he was shocked and in a state of semi-consciousness and was breathing with difficulty. On inspection of his pharynx, I could see fragments of bleeding and swollen tissue hanging down over his laryngo-pharynx, and thought that I might have to perform a tracheotomy. After I had given him one quarter of a grain of morphine and sprayed his pharynx with "Xylocaine" and epinephrine, the relief was immediate and the patient travelled well to hospital. During the trip his pharynx was kept continuously sucked out with an "E.N.J." resuscitator, and he was given oxygen continuously through an intranasal catheter.

In this instance the owner of the property had his own aircraft, but was unwilling to move the patient because of his condition. I should mention as a matter of interest that this man was 12 miles from the homestead when the accident occurred. He drove his land-rover back to the station and opened and closed seven gates on the way; Pavlov would have made capital of this feat.

For several years I have relentlessly encouraged immunization against tetanus for my patients. I have always had a horror of transporting a tetanus patient in an aircraft. At least one man did not heed my warnings, and on the morning of February 16, 1960, he had his first tetanic spasm at 3 a.m. He had a succession of spasms before I reached him. On arrival, I found a sturdy, robust man in a condition of severe spasticity of his jaws, neck and trunk. There was obvious neck retraction and a mild degree of opisthotonos. Swallowing was impossible—he had recently had a spasm and was very frightened. Treatment prior to transport was directed mainly to sedation and muscle relaxation, the correction of dehydration and the commencement of antibiotic therapy. One litre of isotonic glucose-saline solution was set up as an "intravenous drip". Through the tube the patient was given 100 mg. of pethidine, 100 mg. of chlor-

promazine, and 10 ml. of a 10% solution of mephensin. He was also given 500 mg. of oxytetracycline intravenously and 6 grains of "Luminal" intramuscularly. In approximately half an hour the patient was asleep, quite relaxed and ready for transportation. He was then given 50,000 units of antitetanic serum intravenously and 50,000 units intramuscularly, and 1,000,000 units of crystalline penicillin. I did not perform a tracheotomy prior to the flight. The return trip to Charleville was hot and turbulent, and of course there was no shortage of noise and vibration. It lasted approximately two hours and the patient did not have a single spasm. He survived. It is not within the province of this lecture to describe his management in hospital.

Soon after I joined the service in 1953, I was called to attend a similar case. The patient was younger, and on that occasion I did not move the patient to hospital. How different, even in seven years, is our approach to the management of tetanus! On the later occasion I had drugs at my disposal which were unknown to me in 1953.

In the same month we were faced with the transportation of four patients with severe *Salmonella paratyphoid B* food poisoning, all of whom were dehydrated and required stretcher transportation. Fortunately we can just accommodate four stretcher patients in our aircraft. These patients provided an interesting transport problem, since, prior to my arrival, they were having very frequent bowel movements and were suffering from acute spasmodic abdominal pain.

Treatment for each patient prior to transport was as follows: (i) 100 mg. of pethidine; (ii) 50 mg. of chlorpromazine and 0·02 gramme of "Buscopan" given intravenously; (iii) 5 ml. of "Buscopan Composita" given intravenously; (iv) 1 gramme of streptomycin. They were each given 2 oz. of "Kaomycin" orally, and were allowed one ounce of iced water at half-hourly intervals. Not one of these patients complained of abdominal pain on the trip, and there was not a single bowel movement. I believe that the spasmolytic effect of the "Buscopan" was of great help. After their admission to hospital they were placed on intravenous drip therapy. They recovered.

On another occasion I have had three stretcher patients and one sitting patient. The stretcher patients were, first, a women, aged 37 years, who was 28 weeks pregnant and was suffering from acute congestive cardiac failure, which was subsequently proved electrocardiographically to be due to an anterior coronary occlusion. She died after her delivery in Brisbane. The second stretcher patient was a pregnant woman at 36 weeks' gestation, whose Rh antibody titre was rising. She was delivered a few minutes after her admission to hospital. She came into labour during the flight. The third was a young girl, aged 15 years, with acute rheumatism. The fourth patient was a man. This was one of the busiest flights I have ever had. It took me some time to get everything back into my medical bags.

We have had to transfer three patients with coronary occlusion in the past year. The usual treatment prior to flight is the administration of one quarter of a grain of morphine by intramuscular injection, and glycerol trinitrin given sublingually. One of these patients died just prior to landing. When his condition deteriorated, I gave him an amopule of "Pre-Cortisyl" intravenously without effect. The flight should be made at low level, with continuous intranasal administration of oxygen.

Just one year ago, during the course of a routine flight, I stayed overnight at a very isolated station in the far west. The lady of the house had been delivered of a baby just one month prior to this trip, and she was ostensibly very well. At 6 o'clock on the following morning I was awakened by the woman's husband, who told me that she was having a hæmorrhage, and requested me to have a look at her. As I was getting dressed—rather slowly, I might add—he again came to my room and asked if I would see his wife immediately. I then hastened to the patient's bedroom, to find her in a shocked and agitated condition. There was a small pool of blood under the bed, where it had seeped through the mattress and onto the floor. I placed her in the cross-bed position and inserted a Sims speculum. There was a sizeable lump of what appeared to be macerated placenta protruding through the external os, and the bleeding was brisk and the blood bright red. Without anæsthesia, I endeavoured to remove the mass with a sponge forceps (it was firmly adherent and tore with twisting). The bleeding increased in volume, and after catching the anterior lip of the cervix with a volsellum I curetted about one quarter of a pound of placental debris from the uterus, some of which was firmly adherent to the fundal area. After the intravenous injection of 0·5 mg. of ergometrine, the bleeding abated. It would seem that a placenta succenturiata had caused the trouble. When I took her history, the patient confessed to a much smaller hæmorrhage, but of bright red blood, just after her discharge from hospital after her confinement.

I have little doubt that but for the immediate availability of medical aid, this patient would have been *in extremis* in a comparatively short time from uncontrollable uterine hæmorrhage. I doubt that the placental tissue in the os would have been expelled spontaneously. The overnight stop at the homestead was a most fortunate accident.

Patients in congestive cardiac failure frequently require treatment before transport can be undertaken. Some months ago I received a call to a known cardiac sufferer, aged 58 years. He was in acute cardiac failure when we arrived at the homestead. There was every doubt that the patient would have survived the flight without treatment prior to embarkation. Treatment was as follows: (i) the intravenous administration of one quarter of a grain of morphine and one one-hundredth of a grain of atropine; (ii) 0·5 mg. of digoxin given intravenously; (iii) 2 ml. of mersalyl given intramuscularly. During the flight, which was made at 500 feet, oxygen was administered continuously. The patient recovered temporarily.

Cases of dehydration and heat exhaustion are not uncommonly encountered, and the immediate intravenous administration of fluids is a life-saving procedure.

Severe burns are not uncommon, and in the past year we have had three in the Charleville district. One of these victims was a child, aged four years, and is worthy of mention. It is thought that the little girl had been playing with matches. She was found by her mother with her clothes on fire. The flames were put out by rolling her in the dirt. On our arrival, she was given morphine, penicillin and tetanus toxoid and was placed between sterile sheets. She was semi-conscious and very restless. The burnt area was estimated to be 35% to 40% of the body surface, and of this at least 15% was third degree, the remainder being deep second degree. The burns covered most of the front of the trunk (except the upper part of the chest). An intravenous cannula was inserted into the left saphenous vein at the ankle, and she was given 100 ml. (25 grammes) of serum albumin and 400 ml. of normal saline fairly fast. Her condition rapidly improved, and her blood pressure rose from 70/20 to 110/80 mm. of mercury. Her pulse rate fell from 160 to 112 per minute, and she became bright and comfortable. The drip therapy was continued with glucose-saline solution, and she was then ready for transport after the burns had been dressed with "Jelonet". Her subsequent management in hospital demanded the highest in nursing technique, and the child recovered.

Expectant mothers have on innumerable occasions given us unexpected thrills. On two occasions I have had to conduct the confinement on board, and the confusion has to be seen to be believed.

Cases of anoxia from whatever cause are sometimes a problem, particularly if the anoxia is associated with internal hæmorrhage. Here we have the problem of height on the one hand, reducing the partial pressure of oxygen, and the problem of turbulence on the other hand, causing pain and increasing shock. It must be borne in mind that the atmospheric pressure is 25% less at 8000 feet than it is at sea level. The fall in the partial pressure of oxygen can be compensated for only by delivering pure oxygen to the patient up to a certain

height. Thereafter, to be effective, oxygen has to be administered under pressure.

In the preceding paragraphs I have recounted some of the more dramatic and interesting transport problems with which we are confronted; these cases are representative of an average cross-section of our daily practice.

We are all proud of the fact that our organization is alive, progressive and virile; there is no room for complacency. Where next can be spread another fold of Flynn's mantle of safety? Where next can be improved the transportation of the patient? To carry on the banner of our founder we must accept every challenge, and today perhaps the greatest challenge we have seen since 1928 is in taking medical aid to the inhabitants of the Torres Strait islands. Few of us may realize that there are 4000 persons inhabiting the 16 occupied islands of the Torres Straits. These people are in dire need of an aerial medical service, both from the emergency transport point of view and also from the routine medical service aspect. Just two years ago I myself carried out a comprehensive medical survey in this area to determine the need for a service such as we can give. I was assisted by personnel from the Department of Civil Aviation and from both Trans-Australia Airlines and Qantas. We established beyond doubt that, first, a service was a pressing necessity, and also that it was a feasible and practical proposition from the aviation point of view. The proposed base would be at Thursday Island, which has good hospital facilities, and the aircraft would be of the amphibious type.

John Flynn once said: "If you start an idea and it is a good one, nothing can stop it." It is my silent prayer that one day we may be able to extend our service to our less fortunate neighbours off the northern tip of our continent; it is something for which we can all work and plan, and in which pursuit we shall doubtless have the unseen hand of Flynn of the Inland extended to grip that of the helpless and sustain them.

T. J. O'LEARY, C.B.E., M.B., B.Ch.,
Royal Flying Doctor Service of Australia.

British Medical Association.

QUEENSLAND BRANCH: SCIENTIFIC MEETING AT SURFERS' PARADISE.

THE following letter is published at the request of the Assistant Medical Secretary of the New South Wales Branch of the British Medical Association.

British Medical Association House,
88 L'Estrange Terrace,
Kelvin Grove.
5th September, 1961.

Dr. J. G. Hunter,
Medical Secretary,
British Medical Association,
New South Wales Branch,
135 Macquarie Street,
Sydney.

Dear Dr. Hunter,

Our South Coast Local Medical Association is arranging our next Branch Scientific Meeting, in the Moomba Room, Chevron Hotel, Surfers' Paradise, on Friday, 6th October, 1961.

Dr. A. W. Steinbeck, Reader in Medicine at the University of Queensland, will speak on "Clinical Problems of Sex and Intersex".

Refreshments will be served at an informal "get together" starting at 7.30 p.m. before the meeting proper commences at 8.15 p.m.

A cordial invitation to be present is extended to any of your members who may be holidaying on the Gold Coast.

Yours sincerely,
(Signed) JOHN F. LEE,
Honorary Secretary.

MERVYN ARCHDALL MEDICAL MONOGRAPH FUND.

THE following is a second list of donations received for the Mervyn Archdall Medical Monograph Fund. The first list was published in the issue of September 9, 1961.

	£	s.	d.
Dr. G. A. M. Heydon	50	0	0
Mrs. J. M. Vine (Christchurch, New Zealand)	10	0	0
Total	£60	0	0
Previously acknowledged	57	15	0
Total to date	£117	15	0

Out of the Past.

PLAGUE IN QUEENSLAND.[1]

[From the *Australasian Medical Gazette*, May 20, 1903.]

PLAGUE has apparently come to stay. The Commissioner and his assistants are kept busy — at least the latter are — in their efforts to stay its progress. The pendulum has swung back, and the populace are as indifferent now as they were formerly panic-stricken. In order that the Bacteriological Institute may be enabled to devote its time almost exclusively to the requirements of the Commissioner of Health, the latter has caused to be withdrawn the privilege granted by the Home Secretary of free bacteriological examination of specimens obtained from patients suffering from the notifiable diseases and phthisis (excepting leprosy and cholera, which are practically non-existent, and plague which is under the jurisdiction of the Health Department). This will certainly result in a slower and less complete diagnosis being made of such diseases as typhoid and diphtheria by those medical men who have lodge practices and a consequent increase in the spread of those diseases. It may be glorious to be a Commissioner of Health, but for a comparatively new arrival to fly in the face of the practically unanimously expressed opinion of his more experienced fellow-practitioners has the appearance of an arbitrary but futile attempt to bolster up a failing dignity.

Correspondence.

THE ABUSE OF ANTIBIOTICS.

SIR: At the recent Royal North Shore Hospital Reunion Week, one of the things which impressed me most was the danger of the abuse of antibiotics. It seems to me that if we are honest we will agree that we are giving antibiotics at least some of the time — perhaps much of the time — as pure placebos. The cause of this is mostly due to severe pressure of misinformed public opinion. We are well aware that the "mycin" drugs have extremely little effect on virus diseases, and of their grave dangers, but the public are not. Can we not have a campaign to educate them?

I have been giving ascorbic acid, 250 mg. (which has some effect in raising resistance and no dangers) with the statement: "This is the drug we are giving now for virus complaints." It is surprising how the news spreads. I am sure if a number of us adopted it, the pressure to misapply the antibiotics would be broken. Who will join the Anti-Antibiotic Association?

Yours, etc.,

2 Pembroke Street,
Epping, N.S.W.
September 7, 1961.
N. F. BABBAGE.

NOTES ON A THERAPEUTIC COMMUNITY.

SIR: It is not clear just what Dr. Yeomans intends us to understand by his use of the dichotomy "Apollonian" and "Dionysian" in his recent "Notes on a Therapeutic Com-

[1] From the original in the Mitchell Library, Sydney.

munity" (MED. J. AUST., September 2). As I understand the position, Benedict[1] used these two terms (amongst others) to describe some aspects of the *mores* or culture of a relatively small homogeneous group. It hardly seems appropriate to apply such concepts to our heterogeneous population that is busy promoting both immigration and tourism. Such a point of view is expressed by Mead[2] when writing the preface to Benedict's book in 1959 in the following words:

> . . . she [Benedict] was building no typology; she held no belief that Nietzschian or psychiatric labels were suitable for all societies. Nor did she believe that any closed system could be constructed into which all human societies, past, present, and future, would fit.

It would seem that Dr. Yeomans is suggesting that all "alcoholics" belong as a class to the "Dionysian" category and are "impulsive, emotionally labile, and intemperate or lacking a superego". This suggests that all "alcoholics" are what are often called "psychopathic personalities". Further, it seems suggested that all the criminals and other "deviants" are to be similarly considered. From our experience with both "alcoholics" and "criminals", this is a quite untenable proposition, and, however the terms be defined, there is no evidence that we are aware of that "alcoholics" as Dionysians are to be distinguished from the mentally ill Apollonians. Many "alcoholics" are Apollonian (if the term has any real meaning, merit or value), and the research of Cade (1956) as commented upon by Kennedy and Fish[3] is of interest in this context. Cade pointed out that "alcoholism" accounted for about 40% of the male admissions and 8% of the female admissions to a receiving house. The figures for depressive states were virtually the reverse. The conclusion reached was that social and cultural pressures prevent a greater number of women from turning to alcohol when depressed. In any case, "alcoholism" is almost certainly not a simple unitary condition, and many different classifications exist, mostly with many features in common. Thus Syme[4] writes: "There is no warrant for concluding that persons of one type are more likely to become alcoholics than persons of another type"; and Williams[5] describes four main types of "alcoholic"—the "good", the "sad" the "mad", and the "bad". It would seem to consider "alcoholism" as simply "Dionysian" is somewhat naïve and cannot help in either education or research. In a number of tests given to our "alcoholics", the distribution curve has been Gaussian without exception, and there has been no evidence of bi-modality. A similar statement holds good for prisoners. Further, patients in hospital (neurotics) and prison populations tend to have virtually superimposable distribution curves. As an example can be cited the findings with the Maudsley Personality Inventory (Bartholomew[6]; Bartholomew and Marley[7]).

There is, again, no evidence that "criminals" are all "psychopathic" and without a superego. In fact, if Dr. Yeomans was to investigate the matter, he would find that many "criminals" are "normal" people that have had the misfortune to be caught, and that only about 10% to 20% may be termed "abnormal" (Gibbens[8]). Figures of a similar nature are given by many authorities, although some psychiatrists (often analysts) tend to offer more bizarre suggestions (Kraines[9]; Bromberg[10]; Schmideberg[11]; Karpman[12]), but they in turn have been criticized by Glover[13], Neustatter[14] and East[15] for such outrageous claims.

I thought the notion of a prison and a mental hospital being considered as mutually exclusive was no longer really held, particularly by the younger generation of psychiatrists. It is generally conceded today that in very many cases it is utterly fortuitous whether a person ends up in the one or the other. In any case, prison authorities are more and more opening up psychiatric facilities within their own jurisdiction, thus demonstrating that they are aware that the prisoner-patient or Apollonian-Dionysian dichotomy has no meaning or validity. Maddison[16] published an article entitled "A Blue Print for a Model Psychiatric Hospital" that described a situation in 1999. This provoked a letter in the same vein in relation to prisons (Bartholomew[17]). It would seem that Dr. Yeomans has, in certain respects, his feet and mind firmly planted in 1961 rather than in 1999.

Yours, etc.,

4 Adamson Street, Heidelberg, Victoria.
September 10, 1961.

ALLEN A. BARTHOLOMEW.

[1] "Patterns of Culture", 1934, Mentor Books, New York.

[2] Preface to "Patterns of Culture", 1959, Mentor Books, New York.

[3] "Alcoholism, Alcoholic Addiction and Drug Addiction", in "Recent Progress in Psychiatry", 1959, Churchill, London, 3: 277.

[4] *Quart. J. Stud. Alcohol*, 1957, 18: 288.

[5] "Alcoholism", 1956, Livingstone, Edinburgh.

[6] *Brit. J. Delinqu.*, 1959, 10: 120.

[7] *J. ment. Sci.*, 1959, 105: 238.

[8] *Med.-leg. J. (Cambridge)*, 1956, 24: 142.

[9] "Theory of Neuroses and Psychoses", 1943, 2nd Edition, London.

[10] "Crime and the Mind", 1948, Lippincott, Philadelphia.

[11] *Int. J. Psycho-anal.*, 1956, 37: 422; *Brit. J. Delinqu.*, 1956, 7: 44.

[12] *J. crim. Law*, 1956, 47: 8.

[13] *Int. J. Psycho-anal.*, 1956, 37: 311.

[14] *Practitioner*, 1953, 170: 391.

[15] *Med.-leg. J.*, 1944, 12: 69.

NORTHCOTT NEUROLOGICAL DIAGNOSTIC CENTRE.

SIR: A successful post-graduate course in neurology has just been completed at the Northcott Neurological Centre. I am moved to write to your Journal because of a remark made by one of the graduates who attended the course. He asked what kind of place the centre was. On receiving a brief outline of the activities of the clinic, he remarked: "It seems a pity that it is not publicized."

You were kind enough, Sir, to devote space in your columns on September 22, 1951, to a letter by the late Gilbert Phillips, Honorary Consultant Director of the Centre, explaining its objects, etc. Could I remind your readers that this centre was established in 1951 for the investigation and diagnosis of disorders of the central nervous system in ex-servicemen or their dependants? It is excellently equipped to carry out all modern forms of neurological investigation. It is staffed by neurologists, neurosurgeons and specialists in all the ancillary branches of medicine. On one side of the centre is a small private hospital, on the other the rehabilitation clinic of the Multiple Sclerosis Society. It is to be hoped that on this site, one day, a hospital for diseases of the nervous system in the Queen Square manner might arise.

I wish to remind practitioners of the facilities available to their patients at this modern clinic.

Yours, etc.,

K. B. NOAD,
Honorary Director.
Northcott Neurological Diagnostic Centre,
11 Lytton Street,
Cammeray,
New South Wales.
August 24, 1961.

DEMOCRACY.

SIR: The Bazeley affair, and our Federal constitution moves against the wishes of two State Councils, are symptomatic of our time. Pressure groups prevail, responsible elected representation languishes, tensions mount, the species corrupts and bids fair to end itself and all other species.

It has been said men get the government they deserve. I think it is truer that only by indomitable effort will most people gain a voice in their affairs. Class dictatorships are carving up the world. A proletariat party purloins Cuba, the millionaires' mercenaries clamp down on Kuwait and Katanga. As long as most of us are content to leave our affairs to experts and politicians, professional or part-time, just so long we will have the sort of things foisted on us that British public service doctors, but not United States ones, have suffered for about a century.

As a mental hospitals employee I joined and took office in the New South Wales Public Medical Officers' Association. My protests against administrative malpractices were blocked by senior personnel in control of the Association. It may have been in the face of such apathy from his professional association that Dr. Bazeley decided to appeal to the public, not realizing in the urgency of his endeavours that this was against his terms of employment.

It will become incumbent on general practitioners and other groups to form separate organizations, similar to the Public Medical Officers' and specialists' groups, if the

[16] MED. J. AUST., 1960, 1: 33.

[17] MED. J. AUST., 1960, 1: 186.

Australian Association does not speak up for its sections in sectional matters, and above all for public employees who are as individuals publicly muzzled — as our whole fraternity may be ere long.

Grass-roots democratic organization (radical organic democracy) implies a pyramid of power based on functioning committees of ordinary people, retaining power at all levels up to that of a world commonwealth — retaining rights of recall of representatives, rights to referenda (as in Switzerland), and juries to decide on aid to pioneers of ideas that are blocked by academic and bureaucratic hierarchies — just to mention a few points not yet accepted as parts of British justice and the Australian "fair go". As such political participation pervades, society will digest the sick bodies of communist intrigue, capitalist lobbying and bureaucratic corruption to vitalize a cooperative community. This sane bloodless revolution must start in the hearts, minds and deeds of men.

Yours, etc.,

DOUGLAS N. EVERINGHAM.

Post Office Box 328,
Rockhampton,
Queensland.
September 2, 1961.

HÆMOPERICARDIUM COMPLICATING ANTI-COAGULANT THERAPY.

SIR: Dr. John Tziniolis described an interesting case of hæmopericardium complicating anticoagulant therapy in the Journal of August 26. This report underlines the very real danger in using anticoagulants in the presence of an extensive pericarditis due either to the initial myocardial infarction or to the "post-myocardial infarction syndrome" described by Dressler[1] in 1956.

The post-cardiotomy syndrome after heart surgery appears very similar to the post-myocardial infarction syndrome, and I[2] have reported one patient in whom dangerous hæmopericardium resulted from anticoagulants used in the presence of the post-cardiotomy syndrome. It would seem, therefore, that anticoagulants may be contraindicated in the presence of significant pericarditis. If, however, anticoagulants are used advisedly or inadvertently, rigid dosage control is essential. There is now good evidence that Owren's thrombotest (Lempert *et alii*[3], Owren[4]) gives a better indication of a potential bleeding hazard than the one-stage method of Quick.

Yours, etc.,

W. G. SMITH,
Assistant Physician
Superintendent.

Perth Chest Hospital,
Shenton Park, W.A.
September 8, 1961.

THE MANAGEMENT OF MALDESCENDED TESTIS.

SIR: The management of maldescended testes has always been controversial, and the most interesting facet of this problem is that those with the greatest experience in treating this condition still find many points of disagreement among themselves about certain details of management. While agreeing substantially with what Dr. David Dey has said (MED. J. AUST., August 5, 1961), there are certain points with which I disagree. However, I find greater disagreement with the views expressed by Dr. Wyndham in his letter criticizing Dr. Dey's article (MED. J. AUST., September 2, 1961) and therefore think it worth putting a further point of view.

The first point of contention is just what should be meant by the term "ectopic testis". In my view a testis is ectopic if it has a cord of normal length and has descended well beyond the inguinal canal, but cannot be pushed into the scrotum. Therefore I include, as Dr. Wyndham does, the testis which resides habitually in the superficial inguinal pouch, provided that it cannot be made to enter the scrotum. The proviso is important, because the superficial inguinal pouch is a normal structure — in fact, that is where the testis lies in the normal male, whether child or adult, when the scrotum is fully contracted by its dartos muscle. By this definition ectopic testis is much more frequent than Dr. Dey would have it; but I agree with Sir Kenneth Fraser that in such cases one finds a gubernaculum leading towards thigh or perineum or pubis rather than towards scrotum. The presence or absence of a hernial sac has not appeared to me to be diagnostically important, except that a completely patent processus vaginalis does appear to be commoner in undescended than in ectopic testis. I am prepared to admit that it is not always easy to be certain that a testis is ectopic prior to operative exposure, because a number of undescended testes lie in a hernial sac which extends just beyond the external ring, with the ring itself wider than normal. Such a testis, however, is usually impalpable until it is pushed down to the lower end of its hernial sac. I agree with Dr. Wyndham that the testis in childhood is very difficult to palpate if it is within the inguinal canal. A testis which is easily palpated, but is lateral to the external ring, is beyond the canal and lying in the superficial inguinal pouch.

A further point of importance is whether maldescent is unilateral or bilateral. If one testis is of normal size and has been in the scrotum since infancy, it is clear that there has been a normal hormonal urge to descent in intrauterine life or early infancy, and that the testis which is not in the scrotum is either ectopic or has not responded to a normal stimulus. I believe it is true that in many such cases the reason that a testis has not come down is because it is an abnormal testis — a point made by Dr. Wyndham, but a point which to my mind invalidates his previous arguments in favour of the use of hormones in management.

The next point of disagreement concerns the place of hormones in management. There I agree almost entirely with Dr. Dey. Does Dr. Wyndham seriously believe that hormones will produce descent in an established case of non-descent associated with a thick-walled hernial sac which should have disappeared in infancy, and with a firm fascial barrier at the scrotal entrance? And if in such cases "one has often had to repair an inguinal hernia after the successful treatment of maldescent by hormones", would not the patient have been better treated by operation in the first place? I believe that hormones are only for use in the bilateral case in which one has grounds for assuming a deficiency in the natural stimulus to descent, and even in such cases it is unlikely to produce full descent. It may, however, make it possible to put such testes in the scrotum at operation, after removing the hernial sac and other structures tending to shorten the cord.

Dr. Wyndham completely discounts the experimental work on humans which suggests that the longer a testis remains in an extrascrotal position, the less likely it is to attain normal spermatogenesis. In fact, he states that the best time for hormonal treatment is in the eleventh year. With this statement I believe almost all pædiatric surgeons would disagree. The time to correct maldescent of the testis is preferably at the age of five or six, or before that if associated with a clinical hernia, or if demonstrably ectopic. I am inclined to believe that the only testes successfully brought down by hormonal therapy at age eleven are those of the patient with signs of pituitary insufficiency, or of the patient whose retracted testes are wrongly considered to be undescended.

As regards the patient whose testis is not even in the inguinal canal at operation, I agree that in almost all such cases the testis lies at or near the internal ring; moreover, there is usually a small hernial sac in the canal, and when this is pulled down the testis will usually enter the canal. Here is a real surgical problem, because even when the cord is stripped of all restraining structures, including retroperitoneal bands of tissue, it is still quite obviously too short to reach the scrotum. I have not been impressed with the extra length to be achieved by Gross' radical approach to this problem, and further I consider that both this procedure, and the deliberate severing of the short vascular leash as advocated by Dr. Fowler of Melbourne, are liable to lead to atrophy of the testis. Here I am in agreement with Dr. Dey that such testis should be brought down to the region of the external ring and a further operation should be done later. Such later operation usually succeeds, although dissection may be tedious, and the blood supply of the testis is not endangered. As regards the added risk of malignancy in undescended testis, it seems clear that the correction of maldescent does not remove this risk, but it makes it possible to recognize tumour development early, if it should occur. The risk is slight, but sufficiently great to warrant removal of a truly abdominal testis if it cannot be brought outside the abdomen. A testis which can be brought to the external ring, however, should not be removed.

[1] *J. Amer. med. Ass.*, 1956, 160: 1372.
[2] *Lancet*, 1961, 1: 750.
[3] *Lancet*, 1960, 2: 1115.
[4] *Lancet*, 1959, 2: 754.

Finally, as regards the Ombrédanne manœuvre, I agree with both Dr. Dey and Sir Kenneth Fraser. This procedure will always prevent the testis retracting, and it will, in cases where the cord is somewhat short, produce gradual lengthening after operation, without risk of atrophy. I feel sure that any surgeon who has given this method a fair trial will prefer it to any other method of retaining the testis in the desired position post-operatively.

Yours, etc.,

175 Macquarie Street, Sydney, N.S.W.
September 12, 1961.

E. S. STUCKEY.

TETRACYCLINE AND INFANTS' TEETH.

SIR: In the past two years I have, on several occasions, noted yellow discoloration of the deciduous teeth in babies at a follow-up clinic at a maternity hospital. At first I had considered that this was a result of jaundice during the first week of life. However, six months ago I noted the same discoloration in an 18 months old boy with fibrocystic disease of the pancreas who had been treated from infancy with continuous tetracycline. Since then I have observed this phenomenon in other babies who had received tetracycline for as short a period as one week immediately after birth. Not only were the teeth yellow, but the first and second molars were deformed and had extremely sharp cusps. It is not yet known whether the permanent teeth will be affected or not.

In view of these observations, it may be advisable to use other, equally effective, antibiotics when treating small babies.

Yours, etc.,

Shell House,
205 St. George's Terrace,
Perth, W.A.
September 12, 1961.

IAN WALLMAN.

Medical Societies.

THORACIC SOCIETY OF AUSTRALIA (N.S.W. BRANCH).

Annual Scientific Meeting.

THE annual scientific meeting of the New South Wales Branch of the Thoracic Society of Australia will be held on Monday, October 16, 1961, at 8 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney. Dr. D. I. J. Halmagyi, Adolph Basser Fellow of The Royal Australasian College of Physicians in the Department of Medicine, University of Sydney, will speak on "The Inhalation of Body Fluids in the Experimental Animal: Consequences, Mechanism and Treatment". This subject, which is of great practical importance in a country such as Australia, should be of interest to all medical practitioners, and the Society extends a cordial welcome to all.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course in Recent Advances in Cardio-Vascular Diseases.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in recent advances in cardio-vascular diseases will be held on Saturday and Sunday, October 7 and 8, 1961, in the Maitland Lecture Hall, Sydney Hospital. The course will be supervised by Dr. G. E. Bauer. The detailed programme is as follows.

Saturday, October 7. Morning session: "Ischæmic Heart Disease", chairman, Dr. F. L. Ritchie; 10 a.m., "Shock in Myocardial Infarction", Dr. D. G. Julian; 10.30 a.m., "Management of Heart Block", Dr. J. G. Richards; 11.15 a.m., panel discussion on recent advances in ischæmic heart disease: moderator, Dr. F. L. Ritchie; panel, Dr. J. B. Hickie, Dr. D. G. Julian, Dr. J. Raftos, Dr. J. G. Richards. Afternoon session: "Hypertension", chairman, Dr. G. E. Bauer; 2 p.m., "Modern Investigations in Hypertensive Patients", Dr. D. Jeremy; 2.30 p.m., "New Hypotensive Agents", Dr. R. G. Lewis; 3.15 p.m., panel discussion on recent advances in hypertension: moderator, Dr. G. E. Bauer; panel, Dr. R. G. Epps, Dr. D. Jeremy, Dr. R. G. Lewis, Dr. W. A. Seldon.

Sunday, October 8. Morning session: "Valvular and Congenital Heart Disease", chairman, Dr. Douglas Stuckey; 10 a.m., "Recent Diagnostic Methods", Dr. L. Bernstein; 10.30 a.m., "Cardiac Surgery — The Physician's Viewpoint", Dr. E. J. Halliday; 11.15 a.m., panel discussion on recent advances in valvular and congenital heart diseases: moderator, Dr. Douglas Stuckey; panel, Dr. L. Bernstein, Dr. J. F. Farrar, Dr. E. J. Halliday, Mr. H. M. J. Windsor.

The fee for attendance is £3 3s. Those wishing to attend are requested to make early application, enclosing remittance, to the Course Secretary, the Post-Graduate Committee in Medicine, Herford House, 188 Oxford Street, Paddington. Telephone: 31-0671. Telegraphic address: "Postgrad Sydney".

UNIVERSITY OF SYDNEY.

Seminars at the Department of Surgery.

SURGICAL SEMINARS organized by the Department of Surgery, University of Sydney, are held each Tuesday in the Clinical Room, Alfred and Mary Roberts Ward, Royal Prince Alfred Hospital, at 4.15 p.m. Professorial Unit Rounds commence at 2.30 p.m., and post-graduates are welcome to attend. The programme up to the end of October is as follows: September 26, "Malignant Diseases in Childhood", Mr. Douglas Cohen, M.S., F.R.A.C.S.; October 3, "Diverticulitis", Dr. J. May, Surgical Registrar, Royal Prince Alfred Hospital; October 10, "Research Activities in the Department of Surgery", members of the staff of the Department; October 17, "Surgical Emergencies in the New-Born", Mr. J. Steigrad, F.R.A.C.S.; October 24, "Newer Developments in the Surgery of Venous Disorders of the Lower Limb", Mr. Harold Dodd, F.R.C.S., Consulting Surgeon.

SURGICAL SEMINARS AT ST. VINCENT'S HOSPITAL, SYDNEY.

A SURGICAL SEMINAR will be held in the fifth floor Lecture Theatre of the St. Vincent's Hospital, Sydney, on October 9, 1961, at 5.30 p.m. Mr. M. O'Mara will discuss "The Severed Flexor Tendon in the Hand". All medical practitioners are invited to attend this seminar.

INSTITUTE OF CLINICAL PATHOLOGY AND MEDICAL RESEARCH AND LIDCOMBE STATE HOSPITAL.

Seminar Programme.

THE following programme has been arranged for the third term of 1961 for the seminars at the Institute of Clinical Pathology and Medical Research and Lidcombe State Hospital, Lidcombe, New South Wales. Seminars are held in the Lecture Room of the Institute, which is situated in the grounds of Lidcombe State Hospital, on Mondays, commencing at 3.30 p.m. All interested medical practitioners are invited to attend.

October 9, "Army Psychiatry", Dr. H. E. D. Flack, Psychiatrist, Lidcombe State Hospital; October 16, "Histochemical Methods", Dr. K. B. Taylor, Institute of Clinical Pathology; October 23, "Physics in Medical Practice", Mr. W. Caw, C.S.I.R.O., guest speaker, and Dr. T. L. O'Connell, Lidcombe State Hospital; October 30, "Tumours of the Pituitary and the Pituitary Region", Dr. I. J. Hunter, Institute of Clinical Pathology.

November 6, "Management of Old Age", Dr. S. Sax, Lidcombe State Hospital; November 13, "Glycogen Storage Disease", Dr. Ian Thomas, The Royal North Shore Hospital of Sydney, guest speaker; November 20, film session, "Coarctation of the Aorta" and "General Anæsthesia for Dentistry"; November 27, "Some Laboratory Aspects of Cross Infection in Hospitals", with film, Dr. D. Hansman and Miss A. F. Vickery, Institute of Clinical Pathology.

December 4, "Induced Hypothermia", Dr. B. E. Sharkey, Lidcombe State Hospital; December 11, "Overseas Developments in Pathology", Dr. H. Kramer, Institute of Clinical Pathology.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

LIBRARY OF RECORDED LECTURES.

THE following recorded lectures are available on discs from the Melbourne Medical Post-Graduate Committee, 394 Albert Street, East Melbourne. All discs are microgroove and, unless otherwise stated, are ten-inch, the average length of lecture being one hour. Accompanying slides are 2 in. by 2 in. Those wishing to hear them may apply direct to the Committee, or through their B.M.A. Subdivision. Country borrowers have priority. Although their use is not confined to Victoria, it is advised that most of the recordings listed are available from the Post-Graduate Committees in other States, and interstate borrowers are requested to apply to their own centre.

Recorded in 1961.

"Hyponatræmia", by Dr. George Perera, of New York (2 discs); "Diagnostic and Prognostic Evaluation of the Hypertensive Patient", by Dr. George Perera (2 discs); "Cancer of the Colon", symposium conducted by the Melbourne Medical Post-Graduate Committee: nine short lectures by Mr. E. S. R. Hughes, Dr. J. D. Hicks, Dr. P. J. Parsons, Mr. A. R. Kelly, Dr. A. E. Piper, Dr. I. R. Mackay, Mr. S. F. Reid, Mr. Alwynne Rowlands and Dr. E. V. Keogh (8 discs, 204 slides, approximately two and a half hours); "Endocrine Relations", by Professor Douglas Hubble, of Birmingham (2 discs, 19 slides); "Strokes as the Neurosurgeon Sees Them", by Dr. E. Harry Botterell, of Toronto (2 discs, 54 slides); "Cranio-Cerebral Injuries — Prevention and Treatment", by Dr. Botterell (2 discs, 53 slides); "Treatment of Mental Illness in General Hospitals", by Dr. L. G. Kiloh, of Newcastle, United Kingdom (1 disc); "Corticosteroid Therapy in Rheumatoid Arthritis: The Present Position", by Dr. Dudley Hart, of London (2 discs, 24 slides); "Chemotherapy", by Professor W. F. Gaisford, pædiatrician, of Manchester (2 discs, 9 slides).

Recorded in 1960.

"The Reticuloses", a symposium conducted by the Melbourne Medical Post-Graduate Committee: nine short lectures by Professor H. N. Robson, Associate Professor G. S. Christie, Dr. Reginald Motteram, Dr. T. H. Hurley, Dr. J. H. Colebatch, Dr. M. G. F. Donnan and Dr. J. P. Madigan, with discussion opened by Dr. John McLean (6 discs, 140 slides, approximately two and a half hours); "Acute Ischæmia of Limbs", by Professor A. G. R. Lowdon, University of Durham (2 discs, 24 slides); "Hiatus Hernia", by Mr. Norman Tanner, of London (3 discs, 30 slides); "Surgery of Peptic Ulcer", by Mr. Norman Tanner (3 discs, 28 slides); "Recent Advances in Clinical Hæmatology", by Professor William Dameshek, of Boston (3 discs, 81 slides); "Early Diagnosis and Treatment of Cancer of the Rectum and Colon", by Professor J. C. Goligher, of Leeds (2 discs, 38 slides); "Physiological Principles in the Management of Heart Failure", by Professor John McMichael, of London (2 discs, 19 slides); "The Nature of the Staphylococcus Problem", by Dr. Hattie Alexander, of New York (2 discs, 5 slides); "Cancer of the Stomach", a symposium conducted by the Melbourne Medical Post-Graduate Committee: nine short lectures by Professor R. D. Wright, Dr. A. V. Jackson, Dr. R. R. Andrew, Dr. W. Hare, Dr. W. E. King, Dr. L. E. Hurley, Sir William Upjohn, Mr. Grayton Brown and Dr. E. V. Keogh (5 discs, 75 slides, approximately two and a half hours).

Recorded in 1959.

"Cancer of the Cervix Uteri", a symposium conducted by the Melbourne Medical Post-Graduate Committee: nine short lectures by Dr. R. A. Barter, Dr. G. Jacob, Dr. H. F. Bettinger, Mr. J. M. Buchanan, Professor L. Townsend, Mr. A. M. Hill, Dr. G. R. Kurrle, Mr. L. W. Gleadell and Mr. R. Fowler (5 discs, 90 slides, approximately two and a half hours); "Cancer of the Breast", a symposium conducted by the Melbourne Medical Post-Graduate Committee: ten short lectures by Professor S. Sunderland, Professor L. Townsend, Professor E. S. J. King, Professor M. Ewing, Mr. T. Ackland, Dr. W. P. Holman, Dr. H. P. Taft, Mr. Charles Osborn, Mr. Victor Stone and Dr. E. V. Keogh (5 discs, 67 slides, approximately two and a half hours); "Carcinoma of the Thyroid", by Professor R. P. Jepson, of Adelaide (2 discs, 8 slides); "Pain in the Neck and Arm", by Mr. H. Osmond-Clark, of London (2 discs, 75 slides); "Treatment of Diabetes", by Dr. John Nabarro, of London (2 discs, 22 slides); "Management of the Patient with Acute (Virus A) Hepatitis", by Professor Robert Kark, of Chicago (2 discs, 23 slides); "The Nephrotic Syndrome in Adults: A Common Disorder with Many Causes", by Dr. Robert Kark (2 discs, 47 slides); "Emotional Manifestations in Patients with Structural Disease", by Professor R. F. Farquharson, of Toronto (2 discs); "The Value and Dangers of Steroid Therapy", by Professor Farquharson (3 discs, 4 slides); "Medical Diseases of Bone", by Professor Farquharson (3 discs, 24 slides); "Types of Hypertension that may be Relieved by Treatment of a Primary Lesion", by Professor Farquharson (3 discs, 12 slides); "Recent Observations on the Radiology of the Gastro-Œsophageal Junction", by Professor Alan Johnstone, of Leeds (2 discs, 36 slides); "Treatment and Prognosis of Burns", by Mr. W. Gissane, of Birmingham (2 discs, 58 slides); "Clinical Problems of Digitalis Therapy", by Dr. A. Rae Gilchrist, of Edinburgh (2 discs, 15 slides).

Recorded in 1958.

"Diagnosis and Management of the Jaundiced Patient", by Professor Charles Illingworth, of Glasgow (1 disc, 9 slides); "Chronic Pyelonephritis", by Professor M. L. Rosenheim, of London (2 discs, 13 slides); "The Epidemiology of Heart Disease", by Dr. Paul White, of Boston (2 discs, 25 slides); "Metabolic Diseases of Bone", by Dr. M. D. Milne, of London (2 discs, 17 slides); "The Anæmias, their Pathogenesis and Management", by Dr. M. M. Wintrobe, of Salt Lake City, U.S.A. (2 discs, 29 slides); "Leukæmia and Lymph Node Disorders", by Dr. Wintrobe (2 discs, 35 slides); "Carcinoma of the Stomach", by Professor F. A. R. Stammers, of Birmingham (2 discs, 14 slides); "Pain in the Distribution of the Brachial Plexus", by Professor Stammers (2 discs, 16 slides); "Peripheral Arterial Disease", by Professor Stammers (2 discs, 31 slides); "Complications of Partial Gastrectomy — Immediate and Delayed", by Professor Stammers (2 discs, 16 slides).

SYMPOSIUM ON PROBLEMS OF RESPIRATORY FAILURE AND ITS MANAGEMENT.

Some additions are requested to the statement published in the issue of September 16 concerning the Symposium on Problems of Respiratory Failure and its Management arranged under the ægis of the University Department of Medicine, Royal Melbourne Hospital. Attention is drawn to the rearrangement and extension of the programme to include Thursday afternoon and evening (October 26) and Friday morning (October 27). Copies of the programme will be sent to those who enrol. Those attending should indicate whether dinner is required on the evenings of Thursday, October 26, and Friday, October 27. Inquiries, other than those related to registration, may be directed to Dr. Bryan Gandevia, at the Royal Melbourne Hospital. Telephone: FJ 0266, extension 461.

MATER MISERICORDIÆ HOSPITALS, SOUTH BRISBANE.

CLINICAL WEEK.

A CLINICAL WEEK has been arranged by the Mater Misericordiæ Hospitals, South Brisbane, beginning on October 4, 1961. Standing demonstrations will consist of a series of X-ray films and a pathology demonstration. The programme for the week is as follows.

Wednesday, October 4.

9 a.m., registration; 9.30 a.m., "Surgical and Anæsthetic Management of Small Children", Dr. T. R. Brophy and Dr. D. D. McGuckin, Operating Theatre, Children's Hospital; 10.15 a.m., "Disseminated Sclerosis in Queensland", Dr. P. J. Landy, Auditorium; 11 a.m., "Contact Lenses", Dr. E. F. McGuinness, Auditorium; 11.30 a.m., "The Importance of a Biopsy", Dr. C. I. Wilkinson, Auditorium; 12 noon, "The Problem of Chest Pain", Dr. John Ferguson, Auditorium; 12.30 p.m., "Should Gallstones be Removed?", Dr. H. L. Lusby, Auditorium; 2 p.m., "Staphylococcal Pneumonia in Infancy", Dr. D. Clark Ryan, Auditorium; 2.30 p.m., "The Examination of the New-Born Infant", Dr. G. M. Bourke,

Auditorium; 3 p.m., "Neonatal Skin Disorders", Professor T. J. Rendle-Short, Auditorium; 3.55 p.m., "The Recognition and Management of Vascular Emergencies Encountered in General Practice", Dr. D. P. Sapsford, Auditorium; 4.25 p.m., "The Newer Antibiotics in Surgery", Dr. D. M. Portley, Auditorium; 8 p.m., Inaugural Address by Dr. James Hynes, Chairman of Staff; 8.15 p.m., "High Blood Pressure and Pregnancy", Professor Lance Townsend, guest lecturer, Dr. M. J. Eakin, opener of discussion.

Thursday, October 5.

9.10 a.m., "The Persistent Occipito-Posterior Presentation", Dr. I. Cary, Auditorium; 9.40 a.m., "Some Aspects of Ante-Partum Hæmorrhage", Dr. E. J. Esler, Auditorium; 10.10 a.m., "Breech Delivery", Dr. C. J. Murphy, Auditorium; 10.40 a.m., "The Place of Pudendal Block in Obstetrics", Dr. G. J. Carroll, Auditorium; 11.30 a.m., rounds with Professor Townsend at Mater Mothers' Hospital; 2 p.m., "The Surgical Treatment of Œsophageal Pouches", Dr. John Lynch, Auditorium; 2.30 p.m., "Cytotoxic Drugs", Dr. L. I. Burt, Auditorium; 3 p.m., "Religious Stigmatization—Psychiatric Review and Film", Dr. J. V. Hynes, Auditorium; 3.50 p.m., "The Influence of Pregnancy on Cardiac Disorders", Dr. I. R. Ferguson, Auditorium; 4.20 p.m., "Eye Injuries in General Practice", Dr. L. J. Piggott, Auditorium.

Friday, October 6.

9.15 a.m., "Diabetes in Childhood", Dr. N. G. Anderson, Auditorium; 9.45 a.m., "Minor Conditions of the Anus and the Perineum in Children", Dr. D. D. McGuckin, Auditorium; 10.15 a.m., "Some Bladder Conditions in the Female", Urology Department; 11.15 a.m., "Some Gastro-Intestinal Cases of Interest", Dr. D. J. Hodges, Auditorium; 11.45 a.m., "Some Recent Advances in Drug Therapy", Dr. W. S. Rowe, Auditorium; 12.15 p.m., "The Pathology of a Recent Series of Thyroidectomies", Dr. K. O'Reilly, Auditorium; 2 p.m., "Obstetric Aspects of Congenital Anomalies", Professor G. Shedden Adam, Auditorium; 2.45 p.m., "The Surgical Induction of Labour", Dr. R. F. Drake, Auditorium; 3.15 p.m., "Maternal Mortality in Victoria", Professor Lance Townsend, Auditorium; 4.15 p.m., "Amniocentesis in Rhesus Incompatibility", Dr. J. C. Dique, Auditorium; 4.35 p.m., "The Importance of the Investigation of Infertility", Dr. D. J. Dooley, Auditorium.

The afternoon meeting will be chaired by Dr. M. J. Eakin, who will give a short account of the history of the Mater Mothers' Hospital.

Saturday, October 7.

9.15 a.m., demonstration of cases at Adult Out-Patient Department, including orthopædic demonstration.

Sunday, October 8.

8.30 a.m., Mass, Mater Mothers' Hospital Chapel (academic robes); 9.30 a.m., "Functioning Ovarian Tumours", Professor Lance Townsend, Auditorium; 10.30 a.m., Discussion—opener, Dr. Robin Charlton; 11 a.m., "The Surgical Treatment of Parkinsonism", Dr. J. G. Toakley, Auditorium, opener of discussion, Professor Neville Sutton; 11.30 a.m., "A Case of Pheochromocytoma", Dr. J. de Vidas, Dr. T. R. Brophy and Dr. D. P. Sapsford, Auditorium; opener of discussion, Dr. A. W. Steinbeck.

Mater Mothers' Hospital.

Wednesday: rounds, 4 p.m., Dr. M. Eakin, Dr. R. Drake; ante-natal clinic, 1.30 p.m., Dr. M. Eakin, Dr. R. Drake.

Thursday: rounds, 9 a.m., Dr. C. Murphy, 4 p.m., Dr. N. Anderson, Dr. D. Clark Ryan; Out-patients, 1 p.m., Dr. N. Anderson.

Friday: rounds, 11.30 a.m., Dr. D. Dooley, 4 p.m., Dr. I. Cary; ante-natal clinic, 1.30, Dr. D. Dooley, Dr. I. Cary.

University Intelligence.

POST-GRADUATE MEDICAL SCHOOL OF THE AIR.

THE University of New South Wales announces the establishment of the Post-Graduate Medical School of the Air. The University Radio Station, VL2UV, already broadcasts programmes on a wavelength of 1750 kilocycles per second, which is not picked up on ordinary receivers. The adaptation of a standard receiver costs £2 10s.; small transistor sets may offer some difficulties in modification. In addition a registration fee will be payable to the University. Programmes will be circulated in advance, as will illustrated notes giving summaries of talks and symposia. The transmitting area is at present limited to a radius of 20 miles from the University, but the radius will soon be extended to 100 miles.

The Post-Graduate Medical School of the Air will not replace, but should supplement usefully, current methods of continuing medical education. It is often impossible for the doctor in active practice to attend all the medical meetings and read all the journals that he would wish. Once his radio has been adapted he can, without leaving his home or telephone, hear talks by experts including distinguished visitors, panel discussions, reviews of medical literature, etc.; there will be regular times for "Questions and Answers". Tape recordings made overseas by recognized authorities can also be broadcast.

All bodies interested and active in post-graduate medicine will have the opportunity to contribute; they are being invited to nominate representatives on a committee for the Post-Graduate Medical School of the Air. This committee will advise on the subjects to be covered, the selection of speakers, the preparation of notes and other matters. Further information can be obtained from the Executive Officer for Graduate and Extension Studies in Medicine, The University of New South Wales, P.O. Box 1, Kensington.

Australian College of General Practitioners.

QUEENSLAND FACULTY.

Election of Office-Bearers.

THE Queensland Faculty of the Australian College of General Practitioners announces that the following office-bearers have been elected for the year 1961-1962.

Provost: Dr. R. V. Adamson.

Chairman: Dr. Robert Miller.

Vice-Chairman: Dr. B. N. Adsett.

Honorary Secretary: Dr. H. P. Palethorpe.

Honorary Treasurer: Dr. A. J. Parer.

Board Members: Dr. L. A. Little, Dr. Ian Chenoweth, Dr. E. N. Cheesman, Dr. B. C. Harvey, Dr. J. W. P. Henderson, Dr. P. W. Hopkins, Dr. D. O. Jones, Dr. S. A. McDonnell, Dr. H. S. Patterson, Dr. G. L. T. Wright, Dr. L. Fenwick.

Secretary: Mr. R. Spence.

Medical Education Committee: Dr. B. N. Adsett (chairman).

Post-Graduate Subcommittee: Dr. A. J. Parer (chairman), Dr. S. A. McDonnell, Dr. L. A. Little, Dr. R. V. Adamson, Dr. Kurt Aaron, Dr. J. H. Howes.

Graduate Subcommittee: Dr. B. N. Adsett (chairman), Dr. Kurt Aaron, Dr. Owen Powell, Dr. J. W. P. Henderson.

Undergraduate Subcommittee: Dr. E. N. Cheesman (chairman), Dr. D. J. Hodges, Dr. L. Fenwick, Dr. Robert Miller.

Publication Committee: Dr. B. C. Harvey (chairman), Dr. B. N. Adsett, Dr. D. O. Jones.

Research Committee: Dr. G. L. T. Wright (chairman), Dr. Ion Morrison, Professor D. Gordon, Dr. E. H. Derrick, Dr. D. O. Jones.

Preventive Medicine Committee: Dr. H. S. Patterson (chairman), Dr. H. P. Palethorpe, Dr. R. V. Adamson, Dr. R. Patrick, Dr. A. H. Humphry, Professor Rendle-Short, Professor D. Gordon.

The Provost and the Chairman of the Faculty Board are ex-officio members of all committees.

Obituary.

LEONARD JOHN DUNSTONE.

WE are indebted to DR. A. SANDISON for the following account of the career of the late Dr. L. J. Dunstone.

Dr. L. J. Dunstone was educated at LeFevres school and Prince Alfred College, Adelaide, and qualified as a pharmacist at the age of 20 years; because he was too young to practise

pharmacy he switched to medicine, and graduated at Glasgow in 1907. He was a police surgeon in London for a brief period, then returned to South Australia.

He set up practice at Lameroo and established the Lameroo Hospital, which was the first government-subsidized hospital in the Mallee district. After being there for about five years he came to Adelaide and purchased the practice of the late Dr. Morris at Semaphore. On account of his health, he came to live at Woodville, and he remained in active practice there for about thirty years. After that he joined the mental hospitals staff and was acting superintendent at Northfield and Enfield for a few years. He retired at the age of 79 years on account of ill health, and died at the age of 80 years.

During the First World War he served in the Australian forces as captain, and later was transferred to the Navy, where he served for twenty years as District Naval Medical Officer. He was recalled during the Second World War to serve in the same capacity. He was also attached to the St. John Ambulance Brigade for many years and attained the office of Serving Brother. He was a foundation member of the Woodville Masonic Lodge, and also a member of both the Zoological Society and the Agricultural Society, a Justice of the Peace, president of the South Australian Operatic Society and foundation president of the Ex-Naval Men's Association.

Dr. L. J. Dunstone, who died after a protracted illness, will be remembered with affection, not only by his patients, but also by his professional friends. Having been associated with him for many years, I feel able to appreciate fully his well-deserved success. His kindness to his patients helped him, in no small measure, to build up a large practice. Indeed, he was greatly liked by all who sought his help, and for them he worked hard. Our deepest sympathy is extended to his wife and family.

Notes and News.

Seventh International Congress on Diseases of the Chest.

In the issue of September 2, 1961, the date of the Seventh International Congress on Diseases of the Chest, to be held in New Delhi, India, was stated as February 20 to 24, 1964. Word has been received from Mr. Murray Kornfeld, Executive Director of the American College of Chest Physicians, that this date was wrongly stated in the Press release. The Congress will be held on February 20 to 24, 1963.

Victorian Experiment with Personal Health Cards.

The attention of Victorians will be drawn this month to the value of voluntarily carrying personal health cards as a safeguard in case of sudden illness, accident or violence. The cards will be issued at the Royal Melbourne Show between September 21 and 30, by the Australian Mutual Provident Society. An official move towards encouraging Australians to carry personal medical record cards was begun in Tasmania earlier this year. The cards there are being issued by the Tasmanian Health Department, and the move was endorsed by the National Health and Medical Research Council.

The Victorian cards are unofficial. Although they are not as full as Tasmania's, they contain blank spaces for the holder's name, address, telephone number and religion and for the address and telephone number of a relative or friend, with whom contact should be made if necessary. Spaces are also provided for the holder's blood group and Rh type, and for notes about whether the holder is subject to diabetes, epilepsy, hæmophilia or any heart condition, whether he is prone to such conditions as fainting or asthma, whether he is allergic to any drugs, and for dates and types of immunizations and injections that the holder has had.

Cholera Increasing in Hong Kong.

The incidence of cholera is steadily increasing in Hong Kong, according to the weekly epidemiological record of the World Health Organization published on September 1, 1961. The territory had been free of the disease since 1947, when there was one imported case. To date the total number of cases is 97, with 12 deaths. The epidemic mainly affects the boat population, particularly along the western coast, but sporadic cases are now occurring among local residents both on the island and on the mainland. Mass inoculation of the population is being carried out, and half has now

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 26, 1961.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	2(2)	..	..	..	..	..	2
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	2	..	..	..	..	..	1	..	3
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	9(8)	9(7)	..	..	..	14	..	32
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	1	..	1	3	1(1)	..	..	..	6
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	119(43)	71(18)	22(3)	38(31)	4(2)	7(2)	1	8	270
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	1	..	..	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	3(3)	..	..	..	..	..	..	3
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	1(1)	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4	1(1)	2(1)	2	..	..	..	..	9
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	19(10)	1	2	7(7)	..	..	1	30
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	9(3)	3(2)	3(1)	1	1(1)	1	..	1	19
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	1(1)	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	32(16)	12(9)	12(5)	7(7)	8(6)	1	..	1	73
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	3	..	..	..	..	..	3
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

been inoculated. Other necessary control measures are being taken and a thorough watch is being kept on all sources of water. Chlorination of the public water supplies is being stepped up. The first two cases of cholera in the current epidemic were identified on August 17. By August 19 there were 20 cases and three deaths. The following week, ending August 26, there were 51 new cases and five deaths, and from August 27 to 30 (half a week only) there were 26 cases and four deaths.

Hospital Waste Disposal.

A system of hospital waste disposal using paper sacks instead of garbage cans has been introduced into several large hospitals in Sydney and Melbourne. Known as "Garbags", the sacks, which can cope with wet and dry refuse, have been developed by the Australian paper industry to suit Australian conditions. "Garbags" are made of two strong plies of heavy kraft paper. The "wet" sacks incorporate a waterproof barrier between the plies. The sacks, labelled "wet" or "dry", are held in position by a simple clamping mechanism to a patented range of rust-proof metal holders, and are sealed and removed for disposal when full. "The "Garbag" system is in full operation at Sydney Hospital and at St. Vincent's Hospital, Darlinghurst. It is also on trial at several other large Sydney hospitals. In Melbourne, the system has been tested at the Royal Women's Hospital for six months.

In a recent statement, the Chairman of the Victorian Hospitals and Charities Commission, Dr. J. H. Lindell, said that many valuable lessons had been learned during the tests, which would be applied in larger trials embracing six hospitals as well as the Royal Women's Hospital. These had been carefully selected to give a cross section of hospital care in Victoria and included a teaching hospital, a special diseases hospital, a country base hospital and a small country district hospital.

Corrigendum.

ON DEPRESSION.

WE are informed that an error has occurred in the article by I. Pierce James entitled "On Depression", published in the issue of the Journal of September 9, 1961. On page 431, the two figures have been transposed; Figure I is actually Figure II, and vice versa. We apologize to Dr. James for this transposition.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Thong, Francis Khin-Yoong, M.B., B.S., 1957 (Univ. Sydney), St. Vincent's Hospital, Darlinghurst.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Giorgio, Antonio, M.B., B.S., 1955 (Univ. Adelaide), 36 Days Road, Croydon Park.

Hoopman, Peter William, M.B., B.S., 1958 (Univ. Adelaide), 57 Godfrey Terrace, Erindale.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association:

Warren, John Robin, M.B., B.S., 1961 (Univ. Adelaide); Derrington, Arnold Ward, M.B., B.S., 1947 (Univ. Adelaide) D.T.M. & H.

Deaths.

THE following deaths have been announced:

FINCKH.—Alfred Edmund Finckh, on September 2, 1961, at Sydney, N.S.W.

BENJAMIN.—Philip Joseph Benjamin, on September 10, 1961, at Sydney, N.S.W.

CUNNINGHAM.—Alan Laurie Cunningham, on September 10, 1961, at Melbourne, Victoria.

SPENCE.—Kenneth Kinross Spence, on September 12, 1961, at Sydney, N.S.W.

Diary for the Month.

SEPTEMBER 26.—New South Wales Branch, B.M.A.: Hospitals Committee.
SEPTEMBER 27.—Victorian Branch, B.M.A.: Branch Meeting of Council.
SEPTEMBER 28.—South Australian Branch, B.M.A.: Scientific Meeting.
SEPTEMBER 28.—New South Wales Branch, B.M.A.: Branch Meeting.
SEPTEMBER 29.—New South Wales Branch, B.M.A.: Annual (1961) Meeting of Delegates of Local Associations with Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): Medical Officers to Sydney City Council. All contract practice appointments in New South Wales. Members are requested to consult the Medical Secretary before undertaking practice in dwellings owned by the Housing Commission.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full data in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: 68-2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £6 *per annum* within Australia and the British Commonwealth of Nations, and £7 10s. *per annum* within America and foreign countries, payable in advance.